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Applicant last name: LUCIO
Offense: 19.03
Offense code: Capital Murder
Trial court case number: 07-CR-885-B-WR
Trial court name: 138th District Court
Trial court number: 320310138
County: Cameron
Trial court ID: 148
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Court of Criminal Appeals Number:

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WRIT I

CLERK'S RECORD

VOLUME 5

The State of Texas

vs.

MELISSA ELIZABETH LUCIO

Mailed to the Court of Criminal Appeals on
MARCH 30, 2011

Aurora De La Garza
Cameron County District Clerk

CHRISTINA TUSA
Deputy Clerk

Filed in the Court of Criminal Appeals, at Austin, Texas
the day of , 2011.

Louise Pearson,
Clerk of Court of Criminal Appeals

Deputy

No. 07-CR-885-B
Writ No. _____

IN THE
138th JUDICIAL DISTRICT COURT OF
CAMERON COUNTY, TEXAS

and

THE TEXAS COURT OF CRIMINAL APPEALS

Ex Parte MELISSA ELIZABETH LUCIO,
Applicant

APPENDIX
TO APPLICATION FOR WRIT OF HABEAS CORPUS
PURSUANT TO SECTION 4 OF ARTICLE 11.071 OF
THE TEXAS CODE OF CRIMINAL PROCEDURE

VOLUME 3 OF 5

LAW OFFICE OF MARGARET SCHMUCKER

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SBN 24030874

ATTORNEY FOR APPLICANT,
MELISSA ELIZABETH LUCIO

Certificate of service.

TAB

12

CURRICULUM VITAE

THOMAS WILLIAM YOUNG, M.D.

PERSONAL DATA:

Birthdate: June 12, 1956

Birthplace: La Mesa, California

PROFESSIONAL POSITION:

Heartland Forensic Pathology, LLC (private practice)
12717 Oakmont Drive
Kansas City, Missouri 64145

ACADEMIC RECORD:

College: Loma Linda University
College of Arts and Sciences
Riverside, California
October 1974 - June 1977

Medical School: Loma Linda University
School of Medicine
Loma Linda, California
September 1977 - November 1980
Bachelor of Science (Human Biology) and
Medical degrees awarded on November 30, 1980

Internship and Residency: Anatomic and Clinical Pathology
Loma Linda University Medical Center and Jerry L. Pettis
Memorial Veterans Administration Hospital
Loma Linda, California
January 1981 - December 1984

Fellowship: Forensic Pathology
Office of the Medical Examiner, Fulton County (Program
affiliated with Emory University, School of Medicine)
Atlanta, Georgia
July 1988 - June 1989

BOARD CERTIFICATION:

Diplomate-National Board of Medical Examiners
January 2, 1982

Diplomate-American Board of Pathology
Anatomic and Clinical Pathology - May 31, 1985
Forensic Pathology - September 25, 1989

MEDICAL LICENSURE:

Missouri—MD108989
Kansas—04-32417
Georgia—030931 (inactive)

PROFESSIONAL ACTIVITIES:

Heartland Forensic Pathology, LLC (private practice)
Kansas City, Missouri
January 2007 -- Present

Jackson County Medical Examiner
Kansas City, Missouri
July 1995 -- December 2006

Medical Examiner, Platte County Missouri
July 1995 -- December 2006

Medical Examiner, Clay County Missouri
April 1996 – December 2006

Medical Examiner, Cass County Missouri
January 2004 -- December 2006

Director, Office of the Jackson County Medical Examiner
Forensic Pathology Training Program
July 2002 – December 2006

Clinical Associate Professor
University of Missouri-Kansas City School of Medicine
September 1997 – December 2006

Forensic Pathologist, National Disaster Medical System
(DMORT - 7)
United States Department of Homeland Security
October 1996 – March 2006

Participated in Federal Disaster Response involving
Korean Airlines Flight 801, Guam
August 1997

Associate Medical Examiner, Fulton County
Atlanta, Georgia
July 1989 - June 1995

Assistant Professor of Pathology,
Emory University School of Medicine
August 1994 - June 1995

Clinical Assistant Professor of Pathology,
Emory University School of Medicine
July 1991 - August 1994

Medical Examiner for Division of Forensic Sciences,
Georgia Bureau of Investigation
Decatur, Georgia
July 1989 - June 1995

Chief, Anatomic Pathology
Ehrling Bergquist Strategic Hospital
Offutt Air Force Base, Nebraska
January 1985 - May 1988

Medical Director, Phase II
Laboratory Technician Training Program
Ehrling Bergquist Strategic Hospital
Offutt Air Force Base, Nebraska
January 1985 - May 1988

Major, Active Duty
United States Air Force Medical Corps
January 1985 - May 1988

PROFESSIONAL MEDICAL SOCIETIES:

Alpha Omega Alpha Honor Medical Society (1980)
American Society of Clinical Pathologists (1983; Fellow 1985)
College of American Pathologists (Fellow 1985)
American Academy of Forensic Sciences (1989; Fellow 2003)
National Association of Medical Examiners (1989)

PUBLICATIONS:

1. Young TW, Thrasher TV. Non-chromaffin Paraganglioma of the Uterus - A Case Report. Arch Pathol Lab Med 106:608-609, 1982.

2. Young TW, Keeney GL, Bull BS. Red Cell Fragmentation in Human Disease (A Light and Scanning Electron Microscope Study). Blood Cells 10:493-501, 1984.

3. Young TW. Reye's syndrome. A Diagnosis Occasionally First Made at Medicolegal Autopsy. Am J Forensic Med Pathol 13:21-27, 1992.

4. Mandsager NT, Young TW. Pain During Sexual Response Due to Bilateral Bartholin Gland Adenomas. A Case Report. J Reprod Med 37:983-985, 1992.

5. Young TW, Pollock DA. Misclassification of Deaths Caused by Cocaine. An Assessment by Survey. Am J Forensic Med Pathol 14:43-47, 1993.

6. Gulino SP, Young TW. Restraint asphyxia [letter]. Am J Forensic Med Pathol 21(4):420, 2000.

7. Garg U, Althahabi R, Amirahmudi V, Brod M, Blanchard C, Young T. Hyaluronidase as a Liquefying Agent for Chemical Analysis of Vitreous Fluid. J Forensic Sci 49 (2):388-391, 2004.

8. Young TW, Wooden SE, Dew PC, Hoff GL, Cai J. The Richard Cory Phenomenon: Suicide and Wealth in Kansas City, Missouri. J Forensic Sci 50(2):443-447, 2005.

9. Rosales CM, Laboy MA, Young T, Garg U. Death of an Infant Involving Guaifenesin. Tox Talk 30(3):9, 2006.

10. Young TW. Examination of Female Pelvic Organs by *en bloc* Resection. In: Spitz WU, editor. Spitz and Fisher's Medicolegal Investigation of Death: Guidelines for the Application of Pathology to Crime Investigation. Springfield, IL: Charles C. Thomas, 2006.

11. Young TW. Catch-22 No More! Twenty-two lessons learned as a chief medical examiner. http://www.heartlandforensic.com/writing/catch-22. August 12, 2007.

12. Young TW, Okoye MI. The Role of the Forensic Pathologist in a Mass Disaster. In: Okoye MI, Wecht CH, editors. Forensic Investigation and Management of Mass Disasters. Tucson, AZ: Lawyers & Judges Publishing Company, Inc, 2007.

13. Rosales CM, Young T, Laster MJ, Eger EI, Garg U. Sevoflurane Concentrations in Blood, Brain and Lung After Sevoflurane-Induced Death. J Forensic Sci 52 (6): 1408-1410, 2007.

14. Young TW. Forensic Science and the Scientific Method. http://www.heartlandforensic.com/writing/forensic-science-and-the-scientific-method. February 13, 2008.

15. Young TW. An Inferential Test for Expert Testimony. http://www.heartlandforensic.com/writing/an-inferential-test-for-expert-testimony. April 5, 2009.

16. Young TW. Is Sherlock Holmes' "Reasoning Backwards" a Reliable Method for Discovering Truth? Analyses of Four Medicolegal Cases. http://www.heartlandforensic.com/writing/is-sherlock-holmes-reasoning-backwards-a-reliable-method-for-discovering-truth. September 14, 2010.

17. Young TW. Attorneys and Judges, You Can Stop the Madness Now. http://www.heartlandforensic.com/writing/attorneys-and-judges-you-can-stop-the-madness-now. September 19, 2010.

18. Young TW. Fatal Bronchial Asthma With Bilateral Lung Collapse. Am J Forensic Med Pathol 31(4):373-375, 2010.

19. Young TW. Do Resuscitation-Related Injuries Kill Infants and Children? [letter] Am J Forensic Med Pathol 31(4):e6, 2010.

ABSTRACTS AND FORMAL PRESENTATIONS:

1. Hanzlick RL and Young TW: Cocaine-related Death Survey. Presented at the 23rd Annual Meeting of the National Association of Medical Examiners, Sanibel Island, Florida, September 1989.

2. Young TW: Reye's syndrome. A Diagnosis Occasionally First Made at Medicolegal Autopsy. Presented at the 24th Annual Meeting of the National Association of Medical Examiners, Denver, Colorado, September 1990.

3. Young TW and Pollock DA: Misclassification of Deaths Caused by Cocaine. An Assessment by Survey. Presented at the 43rd Annual Meeting of the American Academy of Forensic Sciences, Anaheim, California, February 1991.

4. Young TW: The Significance of Pigmented Pulmonary Macrophages in a Forensic Autopsy Population. Presented at the 45th Annual Meeting of the American Academy of Forensic Sciences, Boston, Massachusetts, February 1993.

5. Young TW: Basics of Death Investigation. Presented at the 105th Annual Meeting of the American Academy of Insurance Medicine, Kansas City, Missouri, October 1996.

6. Pattison CP, Marshall BJ, Young TW and Vergara GG: Is Helicobacter pylori the Missing Link for Sudden Infant Death Syndrome (SIDS)? Supplement to Gastroenterology 112:4, A254, 1997. Presented at the 97th Annual Meeting of the American Gastroenterological Association, May 1997.

7. Young TW, Blanchard CC, Brasfield R: Decapitation by Motorized Shoulder Harness, A Case Report. Presented at the 54th Annual Meeting of the American Academy of Forensic Sciences, Atlanta, Georgia, February 2002.

8. Garg U, Althahabi R, Brod M, Young T, Blanchard C: Hyaluronidase as a Liquefying Agent for Chemical Analysis of Vitreous Fluid. Presented at the Annual Meeting of the Society of Forensic Toxicologists, October 2002.

9. Gill TH, Young TW, Brasfield R: Sudden Death From Post-Traumatic Syringomyelia. Presented at the 37th Annual Meeting of the National Association of Medical Examiners, San Jose, California, September 2003.

10. Young TW, Gill TH, Brasfield R, Duffey J: Death by Bezoar? Presented at the 37th Annual Meeting of the National Association of Medical Examiners, San Jose, California, September 2003.

11. Garg U, Frazee SC, Breckenbach B, Kiscoan M, Johnson L, Miller J, Young TW: Interpreting Postmortem Tricyclic Antidepressant Levels in Vitreous Humor. Presented at the Annual Meeting of the Society of Forensic Toxicologists, October 2003.

12. Young TW, Wooden S, Cai J, Hoff GL, Dew PC: The Richard Cory Phenomenon, Suicide and Socioeconomic Status in Kansas City, Missouri. Presented at the 56th Annual Meeting of the American Academy of Forensic Sciences, Dallas, Texas, February 2004.

13. Gill TH, Young TW, Willard MJ, Garg U, and Dasuki M: Fatty Oxidation Disorders and Sudden Unexpected Death in Children. Presented at the 39th Annual Meeting of the National Association of Medical Examiners, Nashville, Tennessee, September 2004.

14. Gill TH and Young TW: Slavemaster.com, The Serial Murders of John E. Robinson, Sr. Presented at the 38th Annual Meeting of the National Association of Medical Examiners, Nashville, Tennessee, September 2004.

15. Young TW, Brasfield R, and Gill TH: Catch-22 No More! Twenty-two Lessons Learned After 10 years as a Chief Medical Examiner. Presented at the 39th Annual Meeting of the National Association of Medical Examiners, Los Angeles, California, October 2005.

16. Young TW: Forensic Science and the Scientific Method. Presented at the 19th Annual Alumni Reunion Meeting, Department of Pathology and Human Anatomy, Loma Linda University School of Medicine, Loma Linda, California, March 2009.

17. Young TW: Pseudostrangulation. Presented at the 62nd Annual Meeting of the American Academy of Forensic Sciences, Seattle, Washington, February 2010.

18. Young TW: Is It Really So Elementary, My Dear Watson? What Every Attorney Should Know About Flawed Forensic Science Reasoning. Presented at the North American Regional Meeting of the International Society of Family Law, Kansas City, Missouri, June 2010.

19. Young TW: Is It Really So Elementary, My Dear Watson? What Every Attorney Should Know About Flawed Forensic Science Reasoning. Presented at the 15th International Conference of the National Child Abuse Defense and Resource Center, Las Vegas, Nevada, August 2010.

December 9, 2010

HEARTLAND FORENSIC PATHOLOGY, LLC
Finding the truth behind the death

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December 29, 2010

Margaret Schmucker, Esq.
512 E 11th St, Ste 205
Austin, TX 78701

Re: TX v. Lucio

Dear Ms. Schmucker:

You asked me to provide a report containing: 1) my opinions made to a reasonable degree of medical certainty that I would have offered had I testified at the trial, 2) an analysis of the opinions offered by Dr. Norma Jean Farley, the autopsy pathologist, and 3) an analysis of the opinions offered by Dr. Jose Kuri, the defense expert at the trial. In accordance with your request, I viewed the following, and I offer the analyses that follow.

Items viewed:

1. A document labeled "Defendant's Story.pdf."
2. The autopsy report on Alvarez, Myriah (*sic*) by Norma Jean Farley, MD.
3. Multiple photographs from the autopsy, both printed and on CD.
4. Photographs of the stairwell and the bottom of the stairwell.
5. The deposition transcript of Norma Jean Farley, MD.
6. The trial testimony transcript of Normal Jean Farley, MD.
7. The trial testimony transcript of Randall Kenneth Nester.
8. The trial testimony transcript of David Mendoza.
9. The trial testimony transcript of Alfredo Vargas, MD.
10. The trial testimony transcript of Jose Kuri, MD.

11. A CD of images from the skeletal survey from Valley Baptist Hospital.

12. Glass slides from the autopsy, viewed on site at the Cameron County Courthouse in Brownsville, TX on December 27, 2010.

Analyses:

I have analyzed and evaluated the items listed above. The reasonable inferences from that evidence have resulted in the opinions that follow. Those opinions are made to a reasonable degree of medical certainty and follow accepted prevailing forensic pathology standards.

I. My opinions regarding the death of Mariah Alvarez

A. The cause of death is **Blunt head injury, delayed effects**. The manner of death is **Accident**. The initiating event was a head impact from a fall down a set of stairs. The subsequent events, manifested by a progression of signs and symptoms, resulted from that impact.

B. Correlation of witness account with autopsy findings

1. Defendant Melissa Lucio alleged that Mariah fell down stairs. She did not know how many stair steps Mariah fell down. The child was awake and alert and she did not seem disoriented. For this reason, defendant Lucio did not seek medical attention.

2. Some children may develop reactive brain swelling to head trauma, and the swelling may worsen over a period of time. The swelling often leads to the onset of signs and symptoms following a *lucid interval*--an interval of time when there are no observable signs and symptoms. This kind of situation is commonly seen with children following accidental head injury. Literature references (These are submitted with this report):

a. Snoek JW, Minderhoud JM, Wilmink JT. Delayed deterioration following mild head injury in children. *Brain* 1984;107:15-36.

b. Bruce DA, Alavi A, Bilaniuk L, Dolinskas C, Obrist W, Uzzell B. Diffuse cerebral swelling following head injuries in children: The syndrome of "malignant brain edema." *J Neurosurg* 1981;54:170-78.

c. Leestma JE. *Forensic Neuropathology, 2nd ed.* Boca Raton, FL: CRC Press, 2009, pp. 521-22.

3. The defendant describes the worsening of the child's brain function in her statement.

a. Later in the evening following the fall, Mariah said she was feeling sick, and the defendant noted that she was pretty warm ("slight fever"). Ongoing brain swelling can lead to symptoms of nausea. Also, disturbances of hypothalamic functioning (the hypothalamus is a portion of the brain) can lead to disturbances in body temperature.

b. Mariah was up half that night tossing and turning.

c. She remained asleep the next day although she continued to kick her legs, toss and turn. She never awoke until she was found to be dead another day later, prior to the 911 call.

C. Further explanation of the autopsy findings.

1. Hypoxic-ischemic encephalopathy

a. When the brain is deprived of the flow of oxygenated blood, brain damage ensues in the form of *hypoxic-ischemic encephalopathy* (HIE). Dr. Farley uses the term, "anoxic-ischemic encephalopathy" in her report, but it is basically the same condition. "Anoxic" means "no oxygen." "Hypoxic" means "little oxygen." "Ischemic" means damage from insufficient blood flow. "Encephalopathy" means brain damage--in this case from little oxygenated blood flow.

b. With brain swelling brought about by trauma ("cerebral edema"--a term used in her report--means brain swelling), the pressure inside the bony skull rises. As this pressure approaches and exceeds the blood pressure, the blood flow through the brain from arteries conducting blood from the heart slows down. This leads to brain damage. The brain damage leads to further swelling, and a vicious cycle ensues, leading to death.

2. Multiple intracranial and soft tissue hemorrhages

a. As the brain deteriorates, proteins from dead brain tissue are released into the bloodstream, causing an activation of blood clotting factors and blood clotting cells in the blood (platelets). This activation is disorderly and chaotic, spreading throughout the blood stream. It results in the consumption of clotting factors and platelets.

b. This condition, known as *disseminated intravascular coagulation* (DIC), leads to both spontaneous hemorrhages and hemorrhages associated with minor trauma. Handling of the child or even the child's own movements (kicking her legs, tossing and turning) can lead to bruises throughout her body.

c. The absence of coagulation factors and platelets combined with the increasing intracranial pressure from brain swelling can lead to the

subarachnoid and subdural hemorrhages seen at autopsy. The hemorrhages in the lungs and right kidney would also not be unexpected.

3. "Dehydration"

   a. Another complication of the brain deterioration is a condition known as *diabetes insipidus*. The hypothalamus in the brain and a portion of the pituitary gland attached to the hypothalamus allows the release of *antidiuretic hormone* (ADH). This hormone causes the kidneys to retain water.

   b. Without ADH, the child will lose water rapidly from her body, causing the "dehydration" findings.

4. Eye pathology

   a. Hemorrhages in the optic nerve sheath are not diagnostic of head injury. See: Mathses E. Retinal and Optic Nerve Sheath Hemorrhages Are Not Pathognomonic of Abusive Head Injury. Proceedings of the American Academy of Forensic Sciences 62nd Annual Scientific Meeting; February, 2010, p. 272. (included with this report). In this case, brain swelling and DIC caused these hemorrhages. The report of this study is included with this report.

   b. The eyes do not have retinal hemorrhages, according to the eye pathology report. Without retinal hemorrhages, a finding of retinal folds and disruptions is of dubious value. This is because postmortem handling and processing of the eyes can introduce such folds and disruptions.

5. The "bite marks"

   a. The lesions identified as bite marks in the right upper back are not bite marks. They are abrasions and are consistent with the fall down the stairs.

   b. Note the photographs in PowerPoint submitted with this report (Brownsville postconviction case.ppt). They are the photographs of the abrasions in the right upper back but zoomed in to show greater detail. These injuries have numerous fine curvilinear striations, and these are particularly obvious in photograph #3. Several are parallel, but several are not. One fine linear abrasion extends for some distance from the other striations (note photograph #5). Smooth tooth cusps are not capable of forming such fine striations, but impacts with roughened surfaces are (see photographs #6 & 7).

   c. Furthermore, a bite involves a pincer-like action where the two opposing sets of teeth (maxilla and mandible) press inward and toward each other. These

leave impressions in the skin that match the cusps of the teeth. These lesions in this case, instead, represent striated abrasions, made by forces passing roughly in the same direction rather than opposite directions. One of the sets of marks does not reveal an opposing set of marks at all. How can only one set of teeth make marks when the same amount of force is caused by both sets of teeth?

d. These abrasions are consistent with the repeated bouncing of the right upper back down several stair edges. The shapes of these abrasions would also be affected not only by the stair edges but also by the projected contours of the child's scapula and ribs beneath the skin and the contours of a child's upper garment (there are no photographs of an upper garment, nor any description).

6. "Older" lesions

a. Several findings represent either older injuries (the healing left humerus fracture, the right foot laceration, the crusted abrasions on the scalp) or items that might not be injuries at all (thinning of the hair).

b. The older injuries are not necessarily the result of child abuse. They may have been from accidents in an active 2-year-old.

c. Several natural scalp conditions may result in hair loss.

d. There are no available witness accounts to explain or to compare with these findings. As such, any surmised explanation for these findings would be speculative.

7. Focal bronchopneumonia and mucous plug

a. I discovered focal bronchopneumonia and a bronchiolar mucous plug in the lung sections I examined. Dr. Farley did not note these findings in her report.

b. These findings are consistent with deteriorating brain function over a couple of days as described by witnesses.

D. In summary, the autopsy findings are entirely consistent with and explained by the account provided by the defendant. There are no inconsistencies.

II. My analysis of the opinions offered by forensic pathologist Norma Jean Farley, MD.

A. Consider carefully Dr. Farley's conclusion at the end of her autopsy report (I added bold type for emphasis):

Conclusion: It is my opinion that Myriah *(sic)* Alvarez, a 2-year-old female child, died as the result of blunt force head trauma. At autopsy, there is evidence of significant blunt force head trauma with multiple contusions and abrasions involving the head, multiple scalp hemorrhages, cerebral edema, and subarachnoid and subdural hemorrhage (blood around the brain and blood between the brain and skull). The findings in the eyes are **also highly suggestive** of non-accidental trauma. Other autopsy findings are listed above and include contusions involving both lungs and the right kidney, a healing fracture of the left humerus and multiple abrasions and contusions to the body. Dehydration is present and confirmed by vitreous electrolytes. The manner of death is homicide.

B. The "mistake of substituting intuition for scientifically defensible interpretation."

1. In his now classic paper, "Classical mistakes in forensic pathology," Dr. Alan Moritz describes the "mistake of substituting intuition for scientifically defensible interpretation" as "one of the most dangerous mistakes in forensic pathology" and "one that is particularly prevalent among experienced forensic pathologists." Please refer to page 303 of the article that I submitted with this report.

2. Dr. Moritz described how a "Sherlock Holmes type of expert" may intuitively decide from certain autopsy findings that foul play must have occurred without recognizing that such findings may be found in circumstances that are not foul play (Dr. Moritz calls them "control cases").

   a. The famous fictional detective, Sherlock Holmes, would characteristically notice subtle clues and derive from them an account of what happened to lead to those clues.

   b. Such inferences do not work in real life because more than one explanation often exists for any set of physical evidence clues. The success of Sherlock Holmes in being correct almost all of the time was because the author, Arthur Conan Doyle, wrote his stories that way.

   c. Forensic doctors make these mistakes when they fail to learn enough information about their cases, instead intuitively determining what must have happened without complete information.

3. Note Dr. Farley's statement above. There is no evidence in her report that she carefully correlated eyewitness accounts with the autopsy findings to see if the accounts explained the findings. There is no evidence that she was aware of or even interested in the witness accounts. She felt that she could look at the autopsy findings and determine from them the past events that led to those findings, just like Sherlock Holmes.

4. Also note the phrase in bold print, "also highly suggestive." This is not a term

that indicates certainty--reasonable medical certainty or any other form of certainty. It is a term that indicates speculation. Her use of the term, "highly," makes it sound as if she is certain, but she cannot be logically or reasonably certain when more than one explanation can exist for any set of physical findings.

5. On the other hand, if she had carefully compared the autopsy findings with the defendant's account, as I have done in the first part of this report, she would have been able to say with certainty whether or not that account is consistent with the autopsy findings. Such an approach is reliable because no witness--no matter how clever or educated--can invent a lie that perfectly matches complex anatomic and physiologic evidence, particularly when he or she does not know or understand that evidence.

6. Furthermore, more than one person other than the defendant witnessed the events that happened to the child. For child abuse to occur in a setting where there is more than one witness, all witnesses would have to participate in a conspiracy, weaving a tale that would explain complex evidence. There is no evidence that Dr. Farley ever considered such factors in offering her opinions allegedly to a reasonable degree of medical certainty.

7. Included with this report is a document demonstrating the logical basis for what I have written above: Putting It All Together. The Logic Behind the Forensic Scientific Method and the Inferential Test.

C. Examples of speculative opinions from Dr. Farley's testimony

1. Page 7 of her deposition transcript: Dr. Farley sees homicidal beating as the only explanation for the child's injuries, without acknowledging that blunt trauma from a wide variety of means--including non-homicidal means--could lead to the injuries.

Q. Could you make a determination of the cause of these abrasions, contusions and the blood excess of blood around the brain?
A. Yes, that's what blunt force head trauma means, means beat about the head or thrown or hit up against an object, basically; beaten.
Q. And so that we can, a jury can understand this how would, how would this come about?
A. Beating. I don't know how else to make it clear. Beating.
Q. With a hand, a fist, a club?
A. Whatever. We don't know for sure what was used to hit the child, but for sure there's bruises and contusions. We don't see things that we would call a pattern abrasion on the head like from a broom where you get parallel lines or something like that. So all we know that they were beaten. They could be a hand, yes. could be a fist or you can actually take a child and beat their head in with something and that's also possible.

2. Pages 8 & 9: Dr. Farley states that the child's spiral fracture of the humerus is "highly suggestive" and "suspicious" for abuse. These terms indicate speculation, not certainty. She also indicates that the injury took place at an uncertain time previous to the death event.

Q. A broken bone, explain to the jury this broken bone how it occurred, if you know?
A. (Nods head). I don't know how it occurred, if it was twisted. There is usually a torsion-type injury where you grab someone and kind of twist the arm. It's called a spiral fracture. We often see it in abused cases in children from someone grabbing and twisting violently the arm. It goes along with all the other findings or finding on this child of all these contusions and abrasions, and, of course, there's same kind of marks on his arm, so there's actually a fracture there. Children are usually very resilient and don't fracture. And this is a particular type spiral fracture that's highly suggestive and suspicious for abuse.
Q. How old a fracture is this?
A. I can't say for sure it is a healing fracture. Children heal with different stages than adults do. In fact, most people know that their kids' bruises and injuries seem to heal faster than an older person. It's a healing one. It's sort of got fibrosis and granulation in the wound. It's probably several days.
Q. So we're talking days, we're not talking months? A. No, not months.
Q. Okay. More than a week?
A. You can't get that precise with these kind of fractures in children.

(Note: My examination of the glass slides indicates that the humerus fracture is in the remodeling bone stage, where osteoid has matured and cartilage has disappeared. This indicates a time period of "weeks" or even "months," not "days.")

3. Pages 10 & 11: Dr. Farley expresses further speculative opinions about the head injury without any correlation to the witness account.

Q. The bruising, the contusions, this broken bone, broken bone didn't cause the death?
A. No.
Q. The cause of the death was the injury to the head?
A. Yes.
Q. And the injury to the head happened within how much time, do you think?
A. It's hard to say. It's acute. The blood that we're seeing in the subdural space doesn't appear to have any fibroblast or pigment I'm talking about. So I say it was acute. The neuropathologist also said acute, which means usually, you know, within a day or so they start to organize after that. But the exact time we don't know. The child would have had symptoms, in my opinion, immediately after sustaining this type of trauma. But the exact time frame in that 24-hour period I can't give you.

4. Pages 11 & 12: Dr. Farley speculates about the nature of the misinterpreted bite marks and how in her opinion they would have had to have been inflicted. She provides no explanations of how teeth can drag across the skin if they are

pressing skin down and pinching skin together by biting. She indicates that the odontologist was unable to match a bite mold from the defendant to the marks left in the child's back, but she does not explain why the abraded character of the mark would make the odontologist unable to either match with or exclude the defendant.

Q. The torso, explain to this jury what you found in the torso?
A. The same. There are numerous contusions and abrasions. There are also bite marks up on the right back. These bite marks have contusions, but they're also, like, dragging. Looking like dragging of the teeth. There's abrasion associated with them, and if I believe they're on the right back. But again multiple, multiple contusions on the trunk. The trunk would be the abdomen, chest and back. Lots of contusions again.
Q. The bite marks these are human bite marks?
A. In my opinion, they are. There's bite marks on the right upper back. Unfortunately, for an odontologist to tell you and match somebody with them, you have to actually have to leave an impression for them to be able to match, too. In this case they're dragged, they're abraded and there was no way to try to match them.
Q. Okay. How much force is required to leave such marks on a child two years of age?
A. This -- this -- these are pretty bad. I mean, they're abrasions with them. If you picture a child biting someone they leave little impressions of teeth. This actually left blood in the soft tissue as well as a scrape on the surface. So it's not like a little kid just kind of biting and leaving a tiny little teeth impression. This left an abrasion like you get if your knee went across the asphalt.

5. Pages 12 & 13: Dr. Farley speculates about the abusive, severe nature of the trauma to cause the bruises to the lung and kidney without explaining the absence of other findings to indicate severe, deep-seated trauma (i.e. rib fractures, deep soft tissue lacerations and contusions).

Q. Explain how I would get bruises to my lung and kidney?
A. Again, that would be consistent with being beat with an object or objects or kicked or stomped or, you know, however you get the beating that's how it would occur.

6. Page 13: Dr. Farley speculates about the alleged beating, even reconstructing past events from autopsy findings just as Sherlock Holmes might do.

Q. The injuries that this child sustained, did it come all of a sudden at one time, the beating, or is this maybe beatings that took place over a period of time?
A. In my opinion, the child did have signs that it had been beaten over a period of time. The cause of death is a blunt force head trauma, and this time whoever beat the child beat it enough to kill it when they hit it in the head.
Q. And death was it instant or lingers or what?
A. I wouldn't know. You can go in a coma. What I'm saying is when it got this kind of

injury there would have been symptoms immediately, signs and symptoms of the trauma. Either they're lethargic like they're trying to fall asleep or they go into a coma, but they wouldn't be acting normally after this type of head injury. Typically, that's what happens, they'll seize a little bit. They'll be lethargic or sleepy then they might seize a little bit. Every kid is a little bit different. They usually slip into a coma or die.

7. Page 14: Dr. Farley speculates about the cause of some "parallel" bruises that she perceives as a patterned injury without providing a credible scientific explanation as to how "a slap or a hit" could even be capable of causing those bruises.

Q. You have a diagram on your autopsy report that shows that there's, looks like some parallel marks on the inner thigh of the left leg.
A. Yes. That's what I was talking about when I said there were these parallel contusions that were patterned that looked like a slap or a hit. That's what I was talking about when you asked me about extremities earlier.

8. Page 17: Dr. Farley audaciously states that the time a person dies is whenever the doctor says it is. There is no reference or acknowledgement of the accounts of the people who were actually present to see when the child was no longer living.

Q. Were you able to determine, roughly, the time of death?
A. Well, the child I think they pronounced actually at the hospital.
Q. Okay. But she had actually died at home?
A. It doesn't matter. They pronounce, wherever they pronounce is when they die.

9. Pages 14 & 15 of the trial testimony trnascript: There is a failure to acknowledge that there are multiple reasons for patchy hair loss in a child besides trauma.

Q. Did you find any contusions or abrasions on the outside of the scalp?
A. They're more difficult to see in the scalp. We could see the abrasions fairly well. Little scabbed areas in the scalp, and the scalp was very thin. It looked like the hair had been pulled, basically, and then there was a little blood scab in that area.
Q. So across the head up in this area, the child appears to be missing sections of hair, is that correct?
A. Yes. It's very thin and then there's these crusted areas where the hair would have been.
Q. Would that be consistent, Doctor, with the child's hair being pulled away?
A. Yes, it would.

10. Pages 16 & 17 of the trial testimony transcript: Dr. Farley speculates as to the nature and severity of the ear lesions.

Q. Now the ears -- did the child suffer any injuries to the ears?
A. Yes. Both tops of the ears -- both of them had contusions on them -- at the top of the ear, like a pinching. Some people "pop" their kids in the ears when they're not listening. But both sides have these contusions, and it's pretty much in the same area.
Q. And that would be consistent with her being hit, or being pinched in the area? Is that correct?
A. Yes. I mean, all of these bruises are consistent with somebody being hit, or being slammed into something.

11. Page 31 of the trial testimony transcript: Dr. Farley discusses dehydration but fails to mention that such findings can be a delayed effect of head injury, implying instead that the dehydration was brought about by abuse.

Q. Did you examine the child's eyes?
A. Yes, I did. They were sunken.
Q. What is that indicative of?
A. Usually, it's indicative of dehydration -- not getting enough fluid. And I did pull the vitreous from the eyes which is the juice that keeps the eyes open and helps nourish the eyes, and it did show that the child was dehydrated from the electrolytes that we pull from the eyes.

12. Page 32 of the trial transcript: Dr. Farley speculates that the eye findings could only be the direct result from trauma from substantial head injury rather than from other factors.

A. We usually hear about retinal hemorrhages, basically, because it goes along with a blunt head force trauma in children. And, so I could already see, basically -- we'll take the base of the skull, and look to see if we can see injury to the eyes. And on this child there was hemorrhage around both of the nerves that come from the eyes. So that means there was something, probably, traumatically wrong with the eyes. So we did remove them. I send those to San Antonio because there's a specialist -- like I'm a forensic pathologist -- and he's an eye pathologist. So I'll send them to him because he'll take photographs of these as well take very thin sections of them, and then give a report to what he sees. More than just looking in and seeing a retinal bleed, which is just part of the eye where you see blood there. He can then see folds in the retina where the retina has detached and folded onto itself which is, again a sign of significant trauma to the child. And he did see this as well as the optic nerve hemorrhage that I had seen at autopsy.

13. Page 34: Dr. Farley rules out bruising due to a fall, while failing to recognize that the bruising can be a delayed effect of even minor head trauma due to a coagulopathy.

Q. Did you find anything on the child -- any type of injuries that would be consistent with a fall, where you would expect to see -- if something was there as a result of the fall?
A. It would depend on what fall your -- where they're falling from, but not bruises all over the body. That's stretching it a lot, for one fall. Maybe if they fell off a house, fell off a significant height more than once. But these are -- all over the body. This isn't a simple fall.

14. Page 37: Dr. Farley offers speculative opinions regarding the onset of the child's symptoms after a head injury (please refer to scientific articles submitted with this report regarding a lucid interval prior to the emergence of symptoms from brain swelling).

Q. Like on this type of injury, how far back would those symptoms had been known to somebody that is watching the child? At least since the inception, or when?
A. It's usually fairly quickly after the fatal blow occurs that they'll start to have the symptoms. And the first symptom is, they're usually, they're tired. They can't keep them awake. That's the lethargy. They just can't get them up -- can't get them awake. They won't eat or drink, usually. And if they do, they vomit it.

III. My analysis of the opinions of defense expert, Dr. Jose Kuri.

A. In spite of the substantial mistakes made by Dr. Farley, the defense counsel at the trial was not able to point out the mistakes or to provide a scientific defense. This was because of problems with the testimony of the scientific expert they used to confront the state's case.

B. There were several problems with Dr. Jose Kuri.

1. Dr. Kuri was not qualified to perform a forensic analysis.

a. He is a neurosurgeon who treats patients with brain injury and disease. This does not give him the expertise to perform the kinds of analyses required for a complex case like this.

b. Such expertise by qualified forensic professionals is available nationwide and was available at the time of the trial.

c. Certifications by the American Board of Forensic Medicine and the American College of Forensic Examiners do not demonstrate competence for performing forensic pathology. Requirements for certification do not include the kind of full-time, supervised training required of physicians who specialize and obtain board certification in forensic pathology. Dr. Kuri after a fashion acknowledges the limitation of such a credential in pages 10 and 11 of the transcript of his testimony:

"And I told you that I have a diploma of the board, but I -- that is not part of -- this is a type of curriculum. But it's not based on that. I am not going to use it. That kind of paper it's like -- and I told you the ink -- the pen without the ink is just paper. It's a credential. My knowledge is in that part that I am going to talk to you about.

d. Dr. Kuri also limited himself to issues regarding the head, rather than performing the kind of global approach to the entire case required by a forensic analysis. According to page 14:

Q. And you're here to testify about the brain injury that this child suffered, and you're here to testify based upon the findings in the deposition as well as the findings in the autopsy report; is that correct?
A. Yes, sir.
Q. You're not here to contradict what Dr. Farley said the cause of death was?
A. No, sir.

Page 17:

His opinion as to the cause of death, Judge, is going to go into the -- the doctor kept -- Dr. Farley kept talking about 24 hours. Dr. Kuri is going to be testifying that he has seen blunt force trauma to the brain causing hemorrhage and death as much as 48 hours. He is going to be testifying that there was no shake in this -- that there is no indication of shake in the brain.
The Court: He didn't see the brain.
Mr. Gillman: No. From the pictures and from the autopsy report -- excuse me -- from the autopsy report he is able to draw, based upon what the testimony was in the deposition, as well as the autopsy report, that he can testify that there was no shaking.

Page 18:

"He is only going to be talking about the brain, Judge, just the brain. We are not talking about any of the other injuries on the body."

2. Dr. Kuri's testimony was incoherent. Much of the testimony recorded in the transcript did not address the questions asked of him. This may have been due to a combination of factors, such as a language barrier or that the expert was hard of hearing.

a. Incoherence, language barrier and being unresponsive to questions illustrated in pages 36, 37 and 38:

Q. Doctor, if there is evidence that on a Thursday afternoon, a child fell down the stairs and a Friday morning a child was vomiting, on Friday evening the child was somewhat lethargic and not opening their eyes very much and started to cramp, had forms of the locked jaw, and Saturday morning the jaws were shut tight, and then Saturday evening -- Saturday afternoon/evening, the child passed, are those symptoms that you would be interested in knowing if you were trying to treat this child prior to death?
A. Well, these are the cases that I've seen. This is not the first case. Those type of cases that I have seen, that they fell, and they become drowsy. I open the skull, and equate the hematoma with some type of edema. So I remove the bone out to avoid the pressure.
Mr. Padilla: Your Honor, the response is totally unresponsive to the question asked, and I object to that.
The Court: He is talking about what he has seen. Repeat your question.
Q. The symptoms that you are looking for, the symptoms that you are needing to get from in the way of history are telling you what? Do they -- do they tell you how far along or to what degree a person's injury that they have sustained?
A. From what you just mentioned?
Q. Yes, sir.
A. And reading the autopsy report, is hemorrhage, edema and pressure in the peduncle, in the brainstem, and hemorrhage in the brainstem. It's okay. But clinically if the patient fell, and the child has some elasticity, and as I mentioned the child has -- they have better circulation in the brain -- the brain can tolerate more pressure. But symptoms, classical symptoms, they call triad, T-R-I-D-A-I (sic, "triad?") symptoms. Vomiting, blurred vision, and headaches. That was the information. In the injury, we have vomiting and drowsiness. Was the child drowsy?
Q. Yes.
A. Okay. That was related to the accident.
Q. Okay.
A. Then the brain is getting worse through time. And then I was talking before about the kind of brain movement. What kind of movement she becomes ridged.You said she begins to cramps. Any information about the movement?
Q. She seems to be tired, lethargic, doesn't respond in talking with people, and then later on she gets jaws tight, and her body seems to tighten up.
A. Okay. that's part of the complication of the previous to death. When they become decerebrated, unresponsive, the prognosis is bad -- absolutely bad.

b. Difficulty with hearing illustrated in pages 65 and 66:

Q. Doctor, you sat here during the testimony of Dr. Farley.
A. I missed in part --
Q. Okay.
A. -- and she didn't talk loud. So the -- if you would be kind to tell me what she said.

c. On page 44, the defense attorney passed the witness after the witness gave a rambling discourse about head anatomy.

3. Portions of the testimony that could be understood were factually incorrect.

   a. Retinal hemorrhages (in spite of the fact that no retinal hemorrhages were found in the child). The explanation of the cause of retinal hemorrhages is fanciful and incorrect. Page 41:

Q. Could the falling down the stairs cause a hemorrhage in the retina, if you know?
A. The hemorrhage in the retina is due to the hemorrhage in the brain that infiltrates through the exit of the optic nerve. It's part of the -- that's the result.

   b. The "bite marks." Pages 82 and 83:

Q. And that the bite marks would also be consistent with severe trauma to the upper right shoulder, correct?
A. The bite?
Q. Yes.
A. It's not a kiss. It's not -- it's not manifestation of love. This is a trauma. It is sad to see that. It is sad to see that.

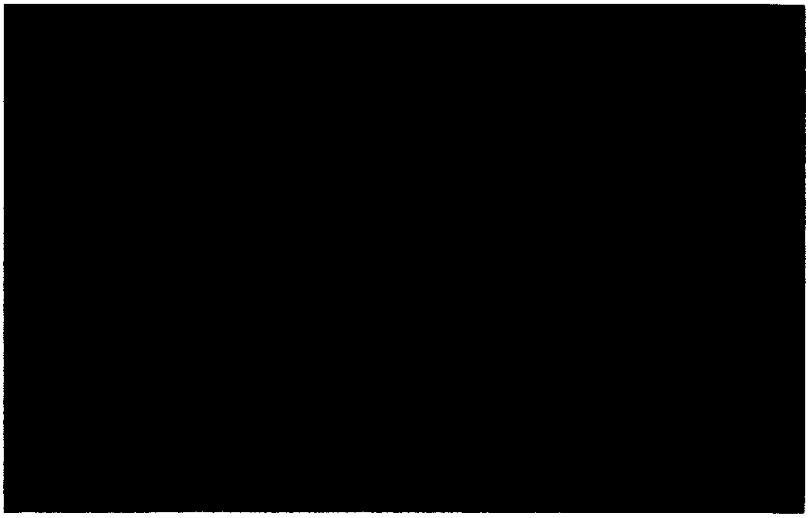
C. In summary, the defense expert in this case was egregiously inadequate to confront the arguments provided by the state for child abuse.

Respectfully submitted,

Thomas W. Young, MD
Heartland Forensic Pathology, LLC

Bite mark?

Bite mark?



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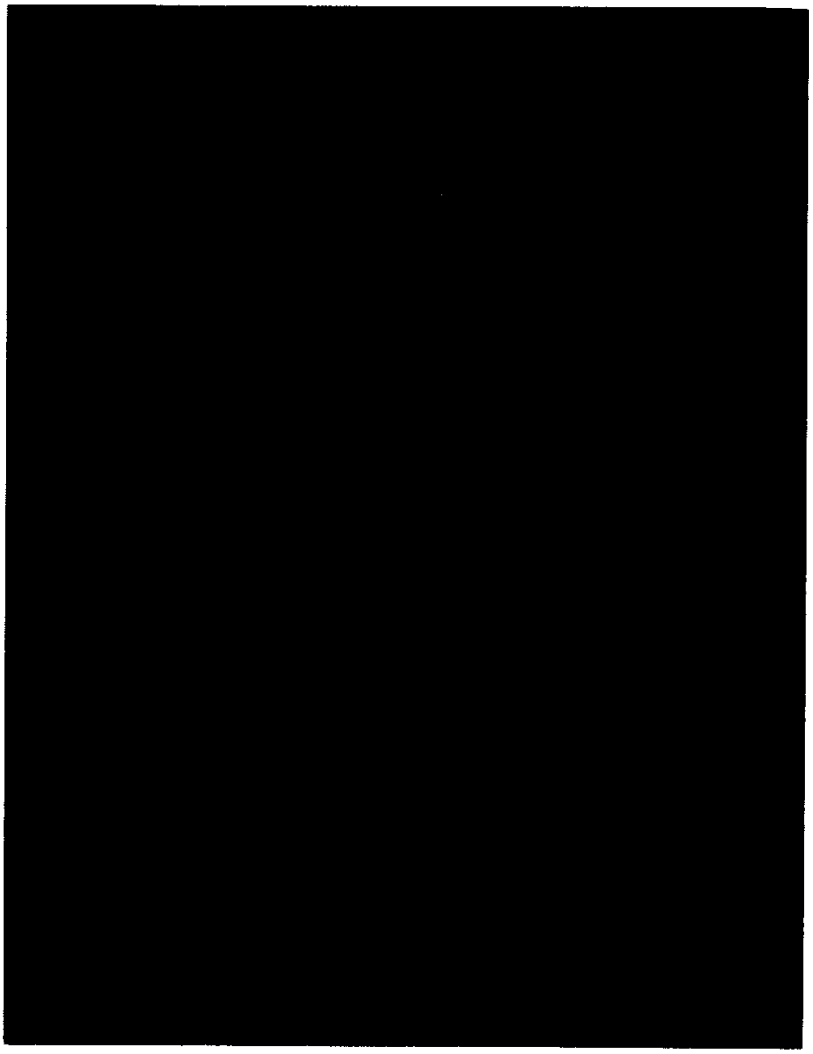

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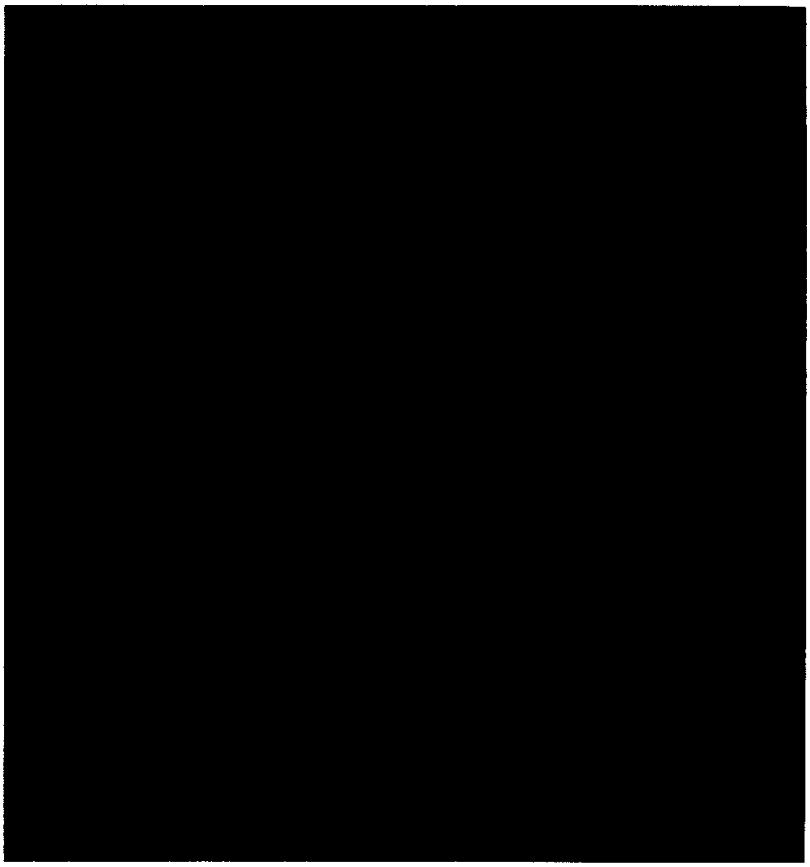

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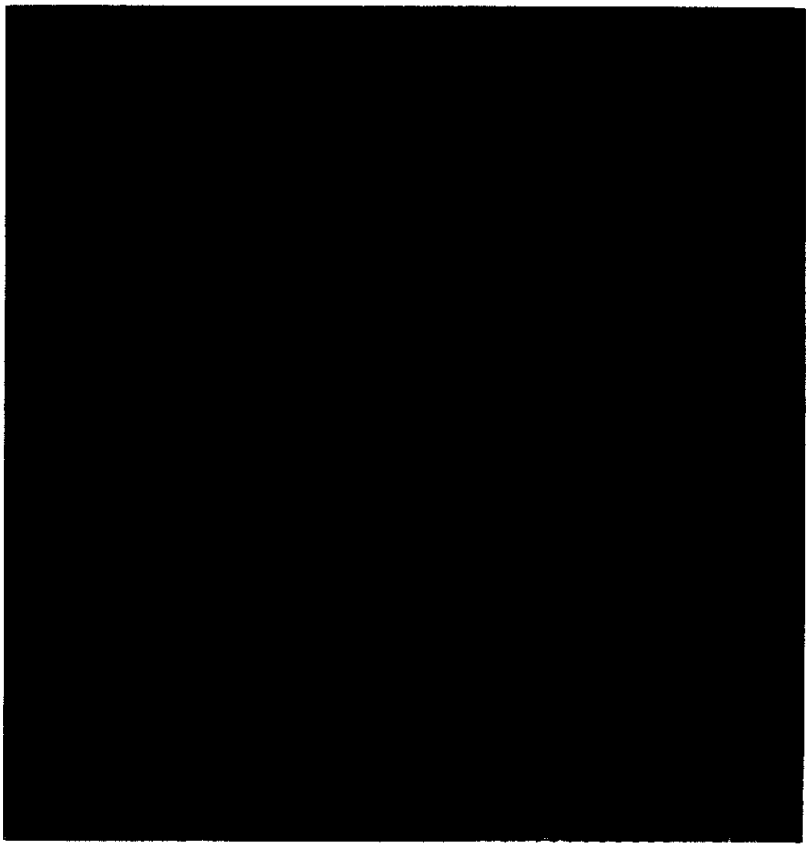

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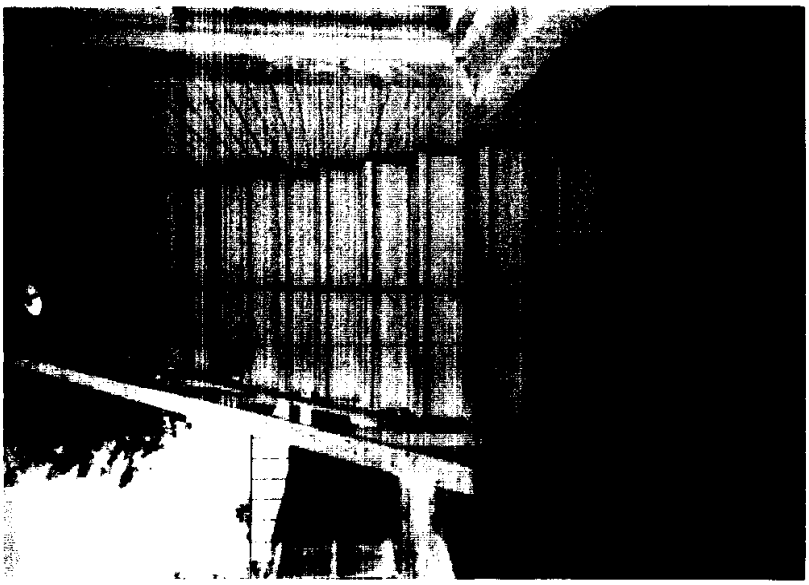
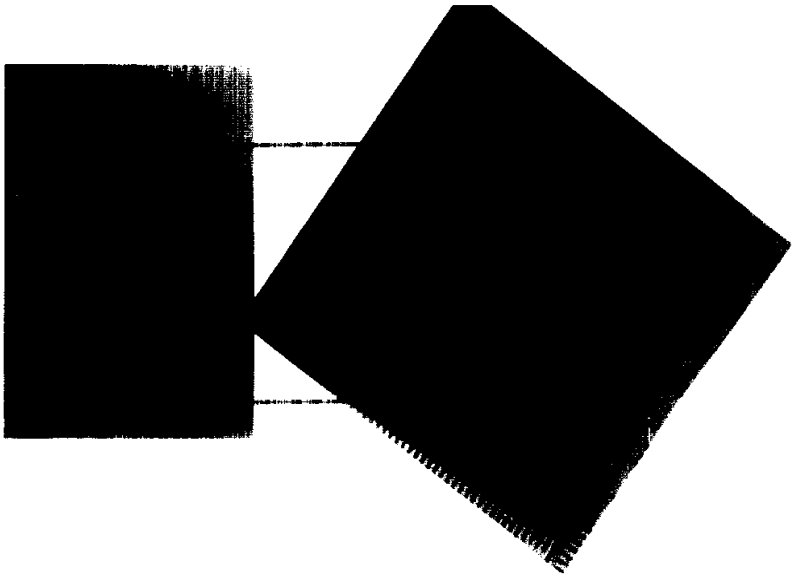
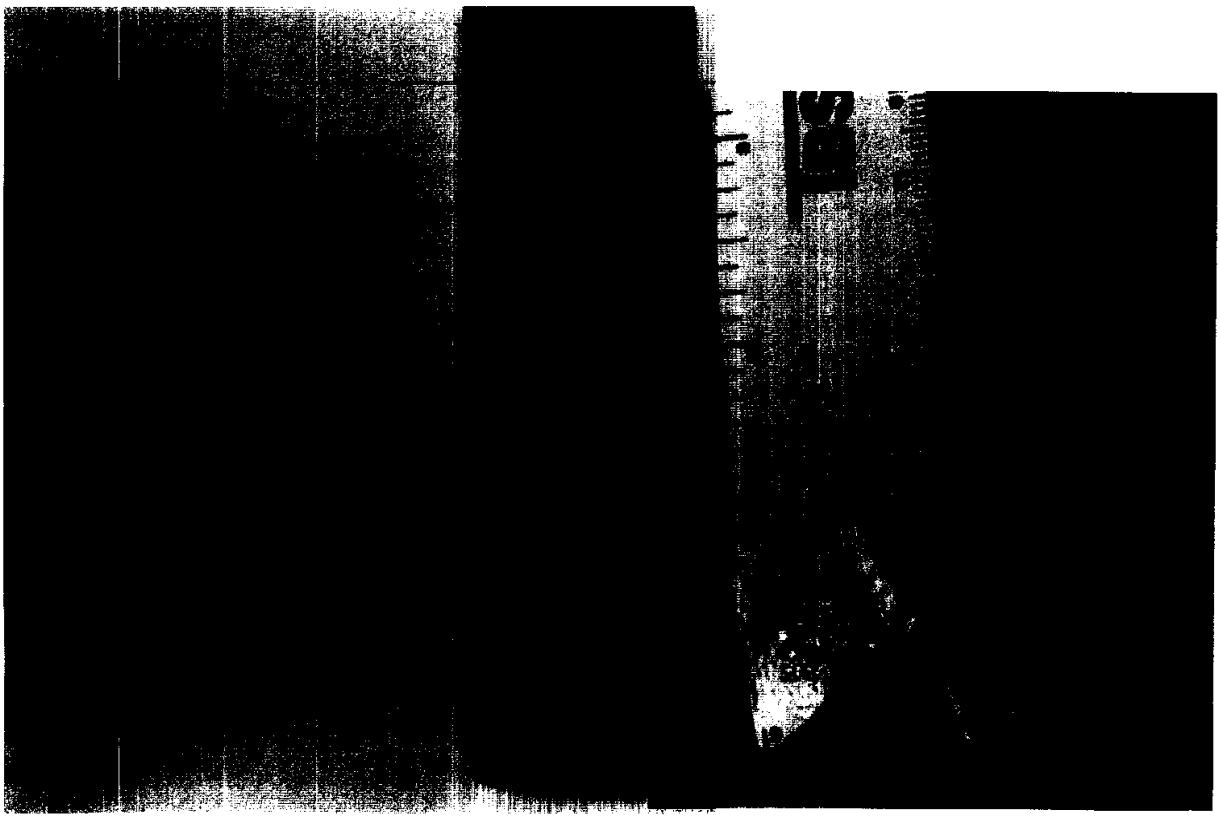
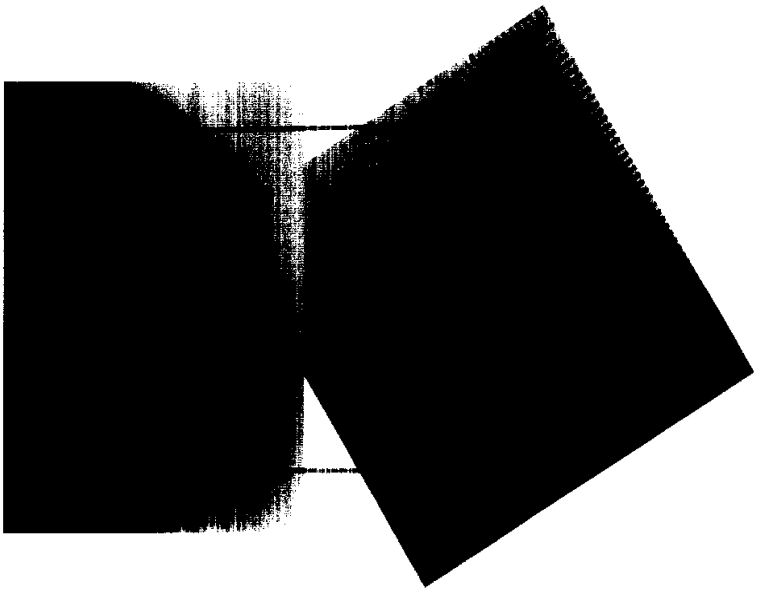


Bottom of stairs







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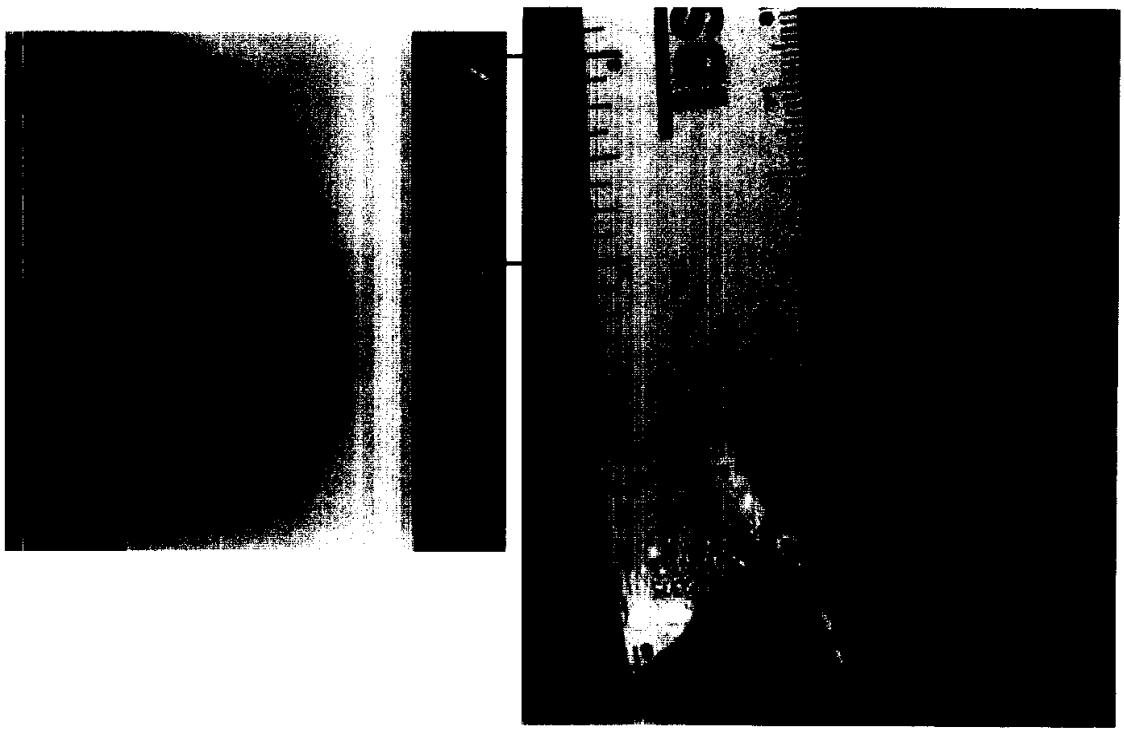

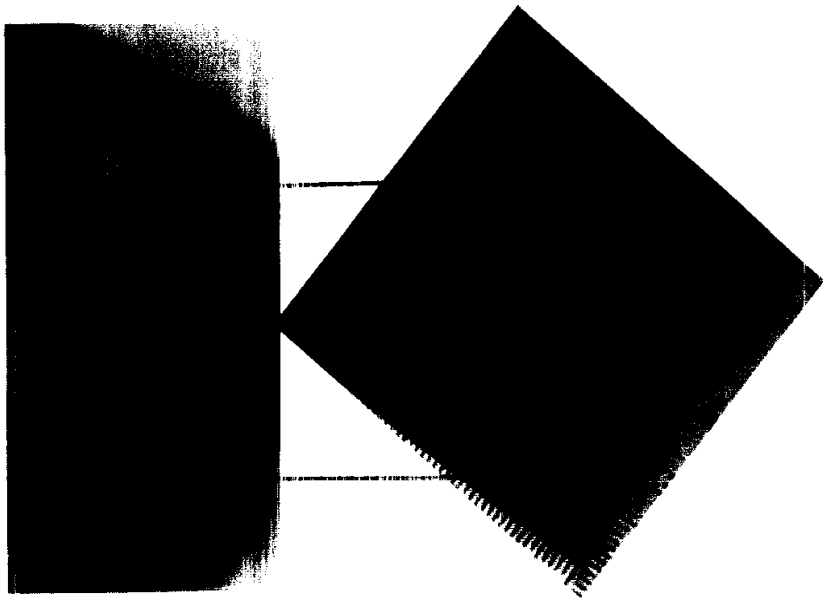

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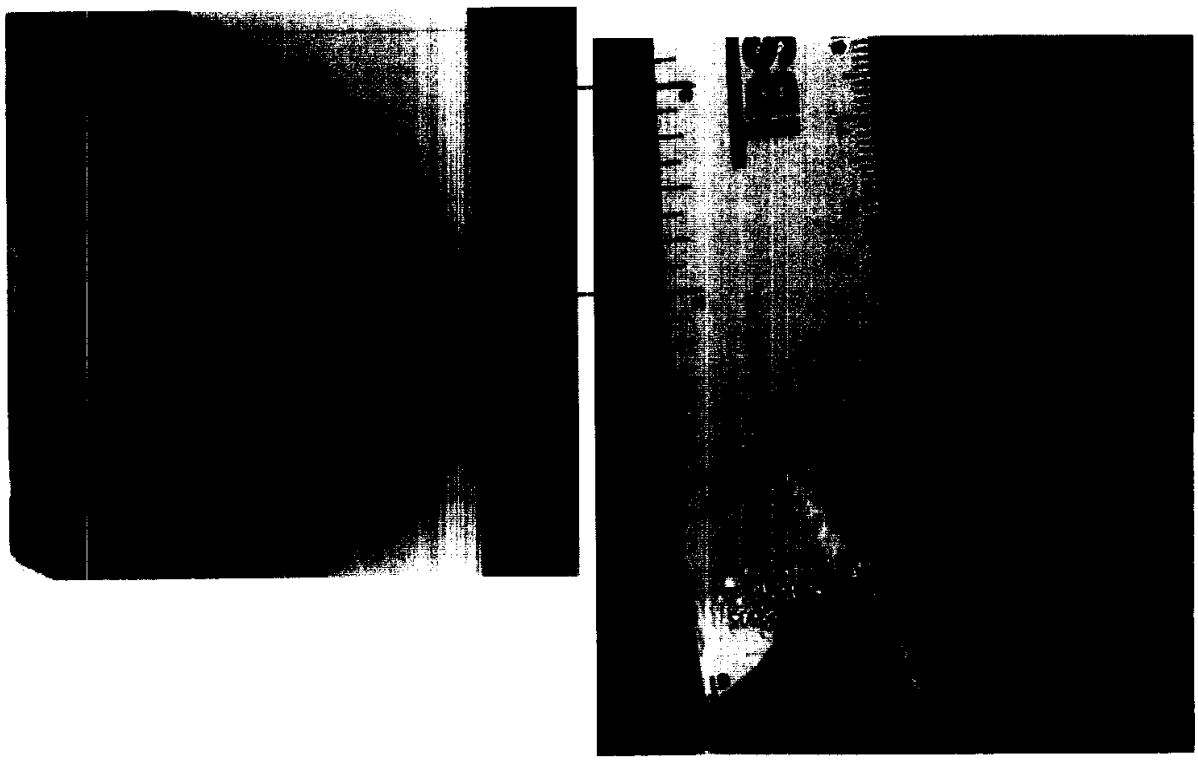

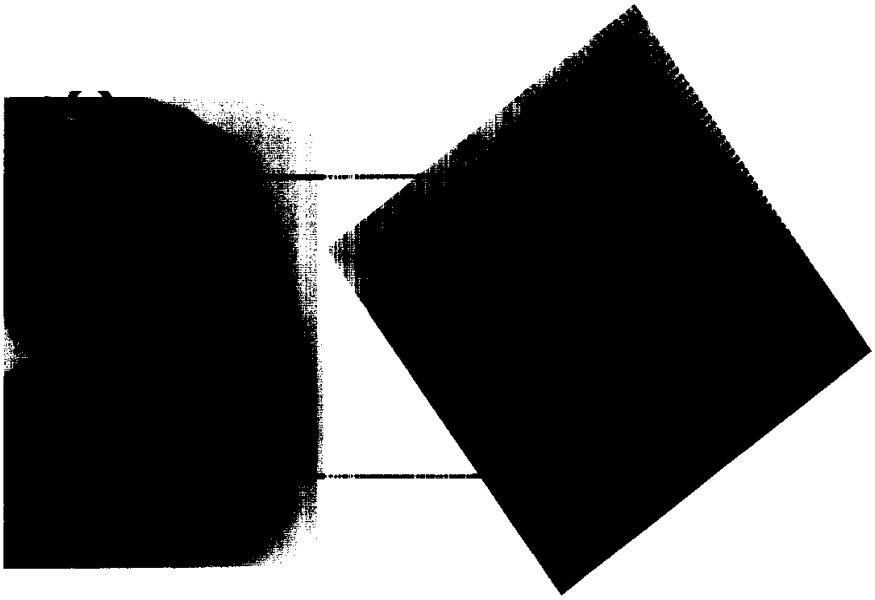

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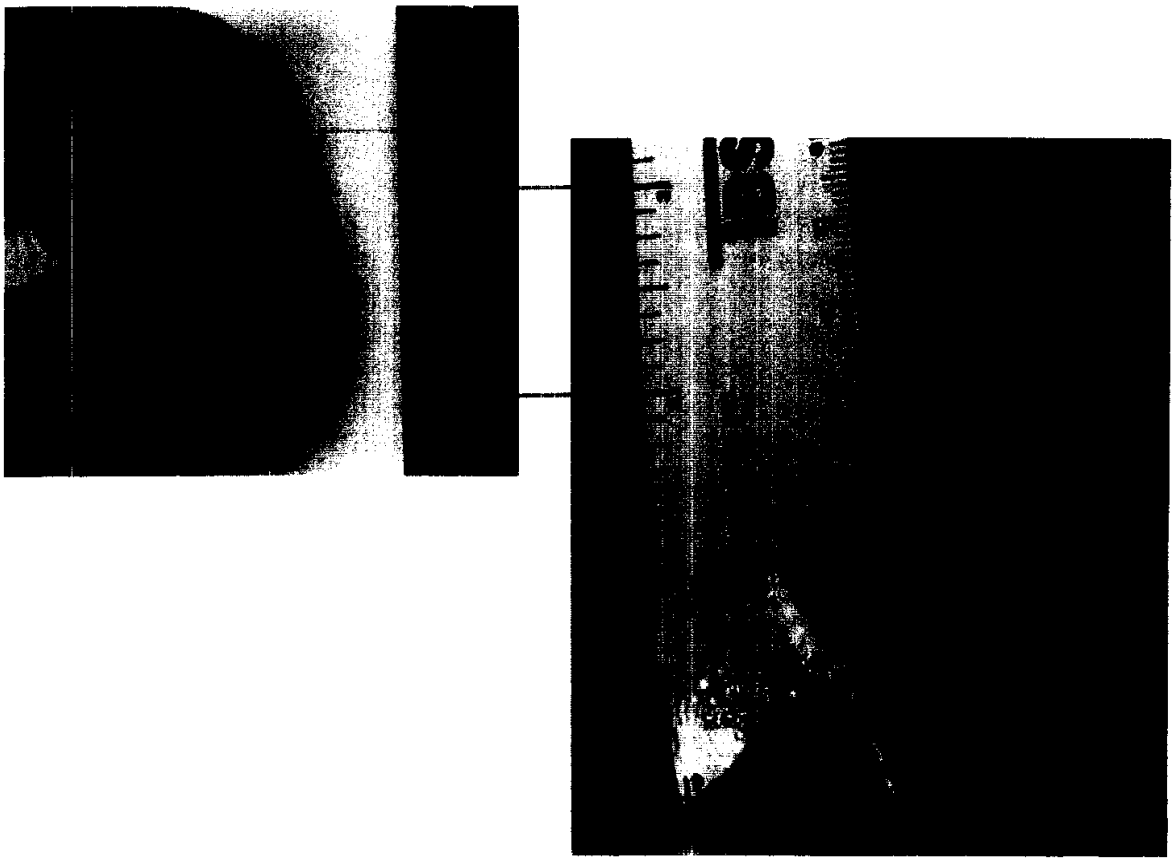

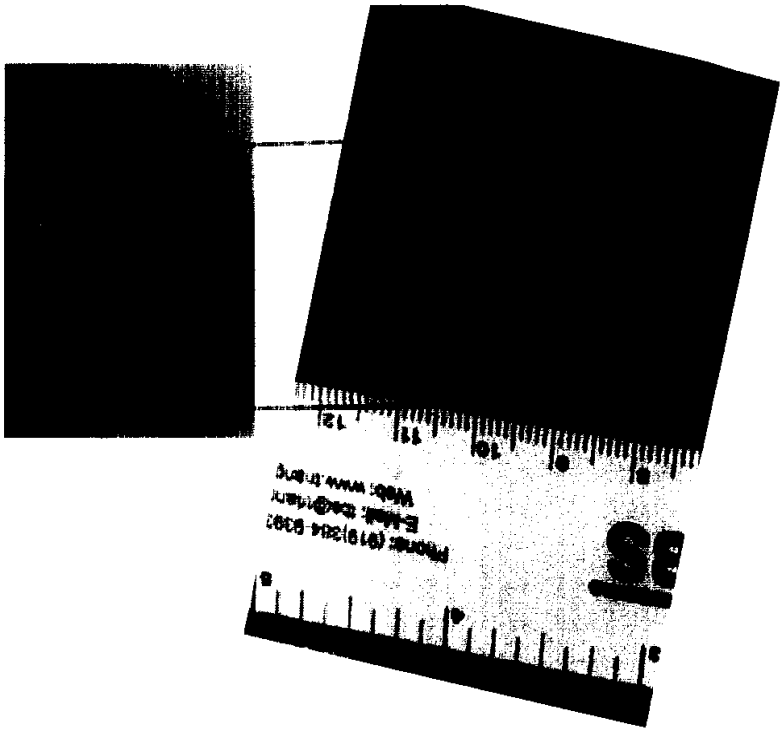

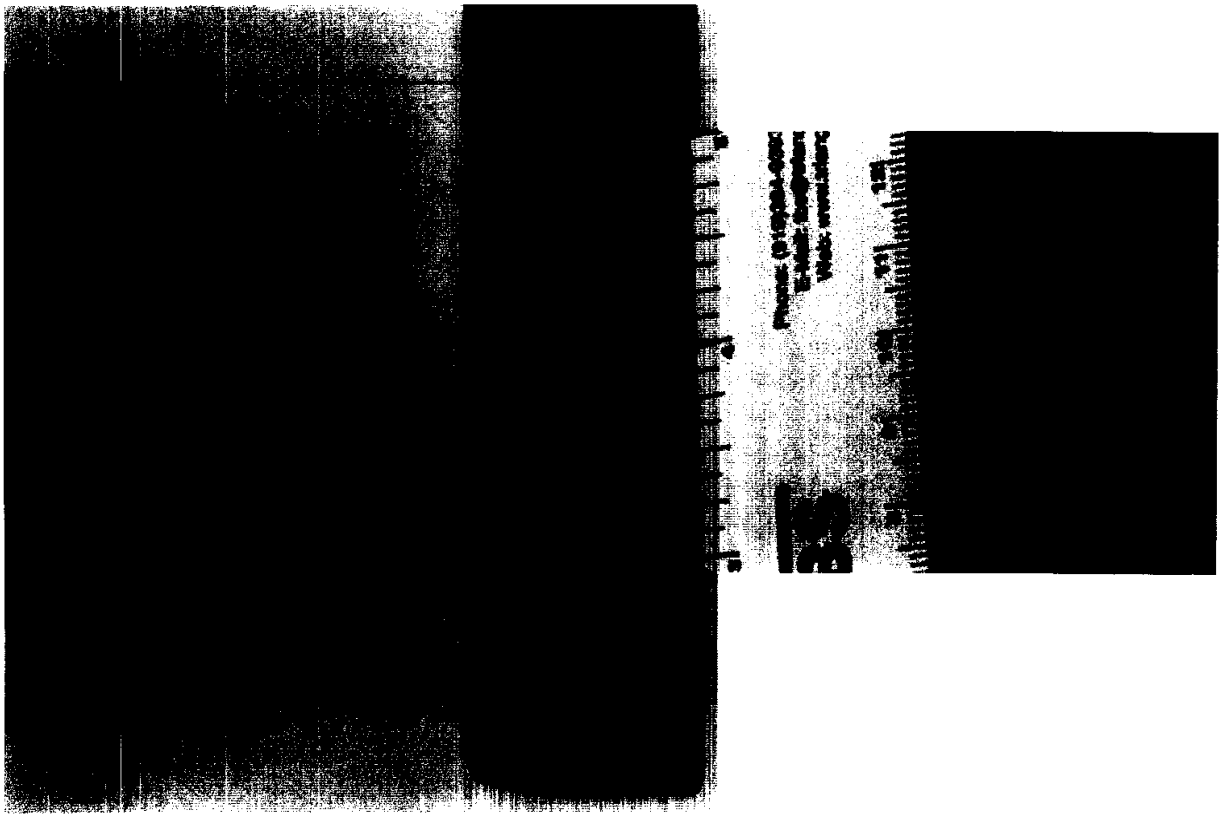

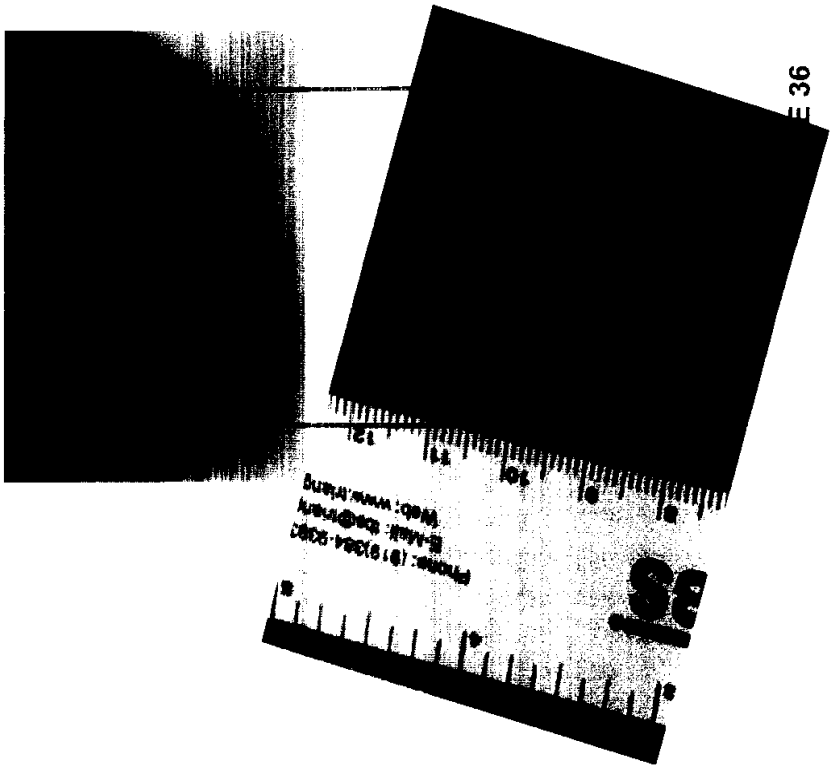

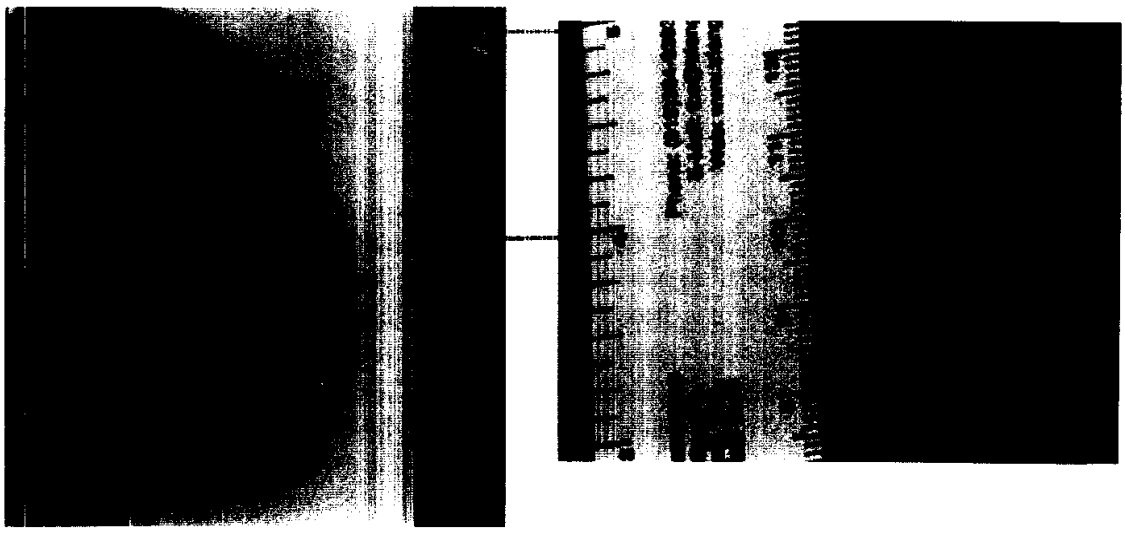

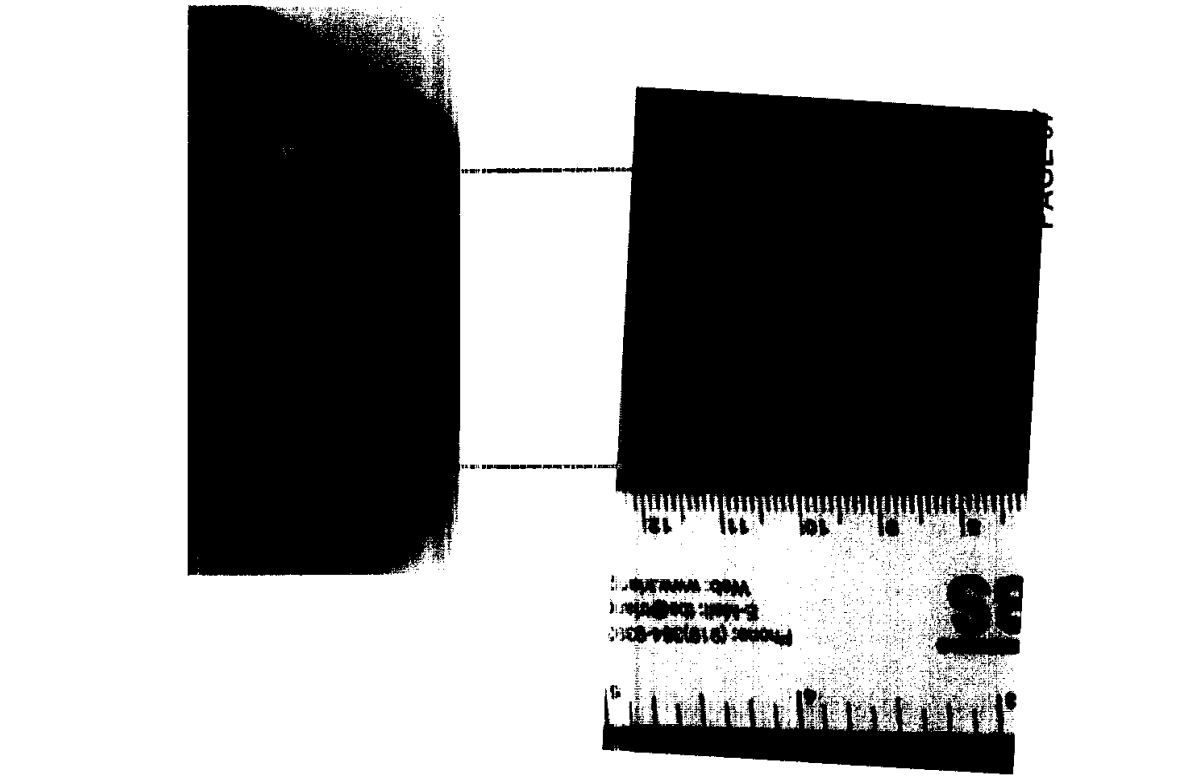

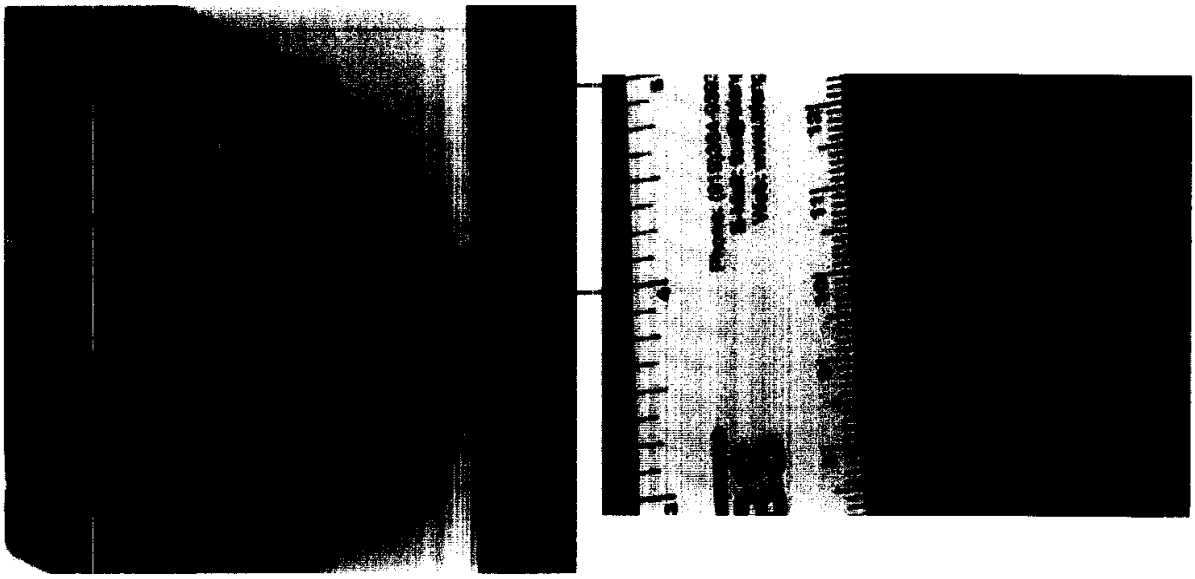

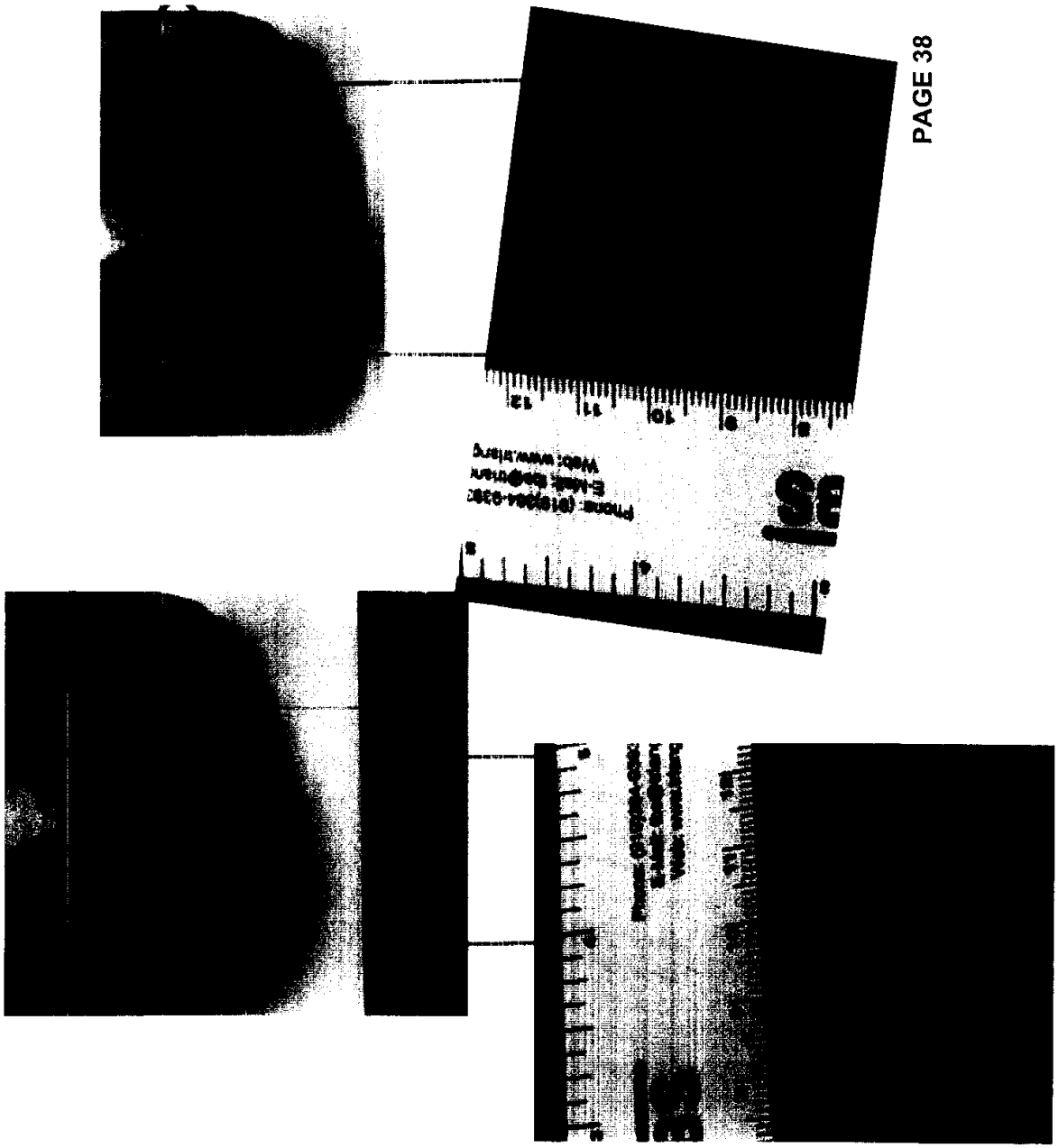

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Fatal Pediatric Head Injuries Caused by Short-Distance Falls

John Plunkett, M.D.

Physicians disagree on several issues regarding head injury in infants and children, including the potential lethality of a short-distance fall, a lucid interval in an ultimately fatal head injury, and the specificity of retinal hemorrhage for inflicted trauma. There is scant objective evidence to resolve these questions, and more information is needed. The objective of this study was to determine whether there are witnessed or investigated fatal short-distance falls that were concluded to be accidental. The author reviewed the January 1, 1988 through June 30, 1999 United States Consumer Product Safety Commission database for head injury associated with the use of playground equipment. The author obtained and reviewed the primary source data (hospital and emergency medical services' records, law enforcement reports, and coroner or medical examiner records) for all fatalities involving a fall.

The results revealed 18 fall-related head injury fatalities in the database. The youngest child was 12 months old, the oldest 13 years. The falls were from 0.6 to 3 meters (2–10 feet). A noncaretaker witnessed 12 of the 18, and 12 had a lucid interval. Four of the six children in whom funduscopic examination was documented in the medical record had bilateral retinal hemorrhage. The author concludes that an infant or child may suffer a fatal head injury from a fall of less than 3 meters (10 feet). The injury may be associated with a lucid interval and bilateral retinal hemorrhage.

Key Words: Child abuse—Head injury—Lucid interval—Retinal hemorrhage—Subdural hematoma.

Many physicians believe that a simple fall cannot cause serious injury or death (1–9), that a lucid interval does not exist in an ultimately fatal pediatric head injury (7–13), and that retinal hemorrhage is highly suggestive if not diagnostic for inflicted trauma (7,12,14–21). However, several have questioned these conclusions or urged caution when interpreting head injury in a child (15,22–28). This controversy exists because most infant injuries occur in the home (29,30), and if there is history of a fall, it is usually not witnessed or is seen only by the caretaker. Objective data are needed to resolve this dispute. It would be helpful if there were a database of fatal falls that were witnessed or wherein medical and law enforcement investigation unequivocally concluded that the death was an accident.

The United States Consumer Product Safety Commission (CPSC) National Injury Information Clearinghouse uses four computerized data sources (31). The National Electronic Injury Surveillance System (NEISS) file collects current injury data associated with 15,000 categories of consumer products from 101 U.S. hospital emergency departments, including 9 pediatric hospitals. The file is a probability sample and is used to estimate the number and types of consumer product–related injuries each year (32). The Death Certificate (DC) file is a demographic summary created by information provided to the CPSC by selected U.S. State Health Departments. The Injury/Potential Injury Incident (IR) file contains summaries, indexed by consumer product, of reports to the CPSC from consumers, medical examiners and coroners (Medical Examiner and Coroner Alert Project [MECAP]), and newspaper accounts of product-related incidents discovered by local or regional CPSC staff (33). The In-Depth Investigations (AI) file contains summaries of investigations performed by CPSC staff based on reports received from the NEISS, DC, or IR files (34). The AI files provide details about the incident from victim and witness interviews, accident reconstruction, and review of law enforce-

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ment, health care facility, and coroner or medical examiner records (if a death occurred).

METHODS

I reviewed the CPSC, DC, IR, and AI files for all head and neck injuries involving playground equipment recorded by the CPSC from January 1, 1988 through June 30, 1999. There are 323 entries in the playground equipment IR file, 262 in the AI file, 47 in the DC file, and more than 75,000 in the NEISS file. All deaths in the NEISS file generated an IR or AI file. If the file indicated that a death had occurred from a fall, I obtained and reviewed each original source record from law enforcement, hospitals, emergency medical services (EMS), and coroner or medical examiner offices except for one autopsy report. However, I discussed the autopsy findings with the pathologist in this case.

RESULTS

There are 114 deaths in the Clearinghouse database, 18 of which were due to head injury from a fall. The following deaths were excluded from this study: those that involved equipment that broke or collapsed, striking a person on the head or neck (41); those in which a person became entangled in the equipment and suffocated or was strangled (45), those that involved equipment or incidents other than playground (6 [including a 13.7-meter fall from a homemade Ferris wheel and a 3-meter fall from a cyclone fence adjacent to a playground]); and falls in which the death was caused exclusively by neck (carotid vessel, airway, or cervical spinal cord) injury (4).

The falls were from horizontal ladders (4), swings (7), stationary platforms (3), a ladder attached to a slide, a "see-saw", a slide, and a retaining wall. Thirteen occurred on a school or public playground, and five occurred at home. The database is not limited to infants and children, but a 13-year-old was the oldest fatality (range, 12 months–13 years; mean, 5.2 years; median, 4.5 years). The distance of the fall, defined as the distance of the closest body part from the ground at the beginning of the fall, could be determined from CPSC or law enforcement reconstruction and actual measurement in 10 cases and was 0.6 to 3.0 meters (mean, 1.3 ± 0.77; median, 0.9). The distance could not be accurately determined in the seven fatalities involving swings and one of the falls from a horizontal ladder, and may have been from as little as 0.6 meters to as much as 2.4 meters. The maximum height for a fall from a swing was assumed to be the highest point of the arc. Twelve of the 18 falls were witnessed by a noncaretaker or were videotaped; 12 of the children had a lucid interval (5 minutes–48 hours); and 4 of the 6 in whom funduscopic examination was performed had bilateral retinal hemorrhage (Table 1).

CASES

Case 1

This 12-month-old was seated on a porch swing between her mother and father when the chain on her mother's side broke and all three fell sideways and backwards 1.5 to 1.8 meters (5–6 feet) onto decorative rocks in front of the porch. The mother fell first, then the child, then her father. It is not known if her father landed on top of her or if she struck only the ground. She was unconscious immediately. EMS was called; she was taken to a local hospital; and was ictal and had decerebrate posturing in the emergency room. She was intubated, hyperventilated, and treated with mannitol. A computed tomography (CT) scan indicated a subgaleal hematoma at the vertex of the skull, a comminuted fracture of the vault, parafalcine subdural hemorrhage, and right parietal subarachnoid hemorrhage. There was also acute cerebral edema with effacement of the right frontal horn and compression of the basal cisterns. She had a cardiopulmonary arrest while the CT scan was being done and could not be resuscitated.

Case 2

A 14-month-old was on a backyard "see-saw" and was being held in place by his grandmother. The grandmother said that she was distracted for a moment and he fell backward, striking the grass-covered ground 0.6 meters (22.5 inches) below the plastic seat. He was conscious but crying, and she carried him into the house. Within 10 to 15 minutes he became lethargic and limp, vomited, and was taken to the local hospital by EMS personnel. He was unconscious but purposefully moving all extremities when evaluated, and results of funduscopic examination were normal. A CT scan indicated an occipital subgaleal hematoma, left-sided cerebral edema with complete obliteration of the left frontal horn, and small punctate hemorrhages in the left frontal lobe. There was no fracture or subdural hematoma. He was treated with mannitol; his level of consciousness rapidly improved; and he was extubated. However, approximately 7 hours after admission he began to have difficulty breathing, both pupils suddenly dilated, and he was rein-

TABLE 1. *Summary of cases*

No.	CPSC No.	Age	Sex	Fall from	Distance M/F	Witnessed	Lucid interval	Retinal hemorrhage	Subdural hemorrhage	Autopsy	Cause of death	FP
1	DC 9108013330	12 mos	F	Swing	1.5–1.8/5.0–6.0	No	No	N/R	Yes +IHF	No	Complex calvarial fracture with edema and contusions	No
2	AI 890208HBC3088	14 mos	M	See-saw	0.6/2.0	No	10–15 minutes	No	No	No	Malignant cerebral edema with herniation	No
3	IR F910368A	17 mos	F	Swing	1.5–1.8/5.0–6.0	No	No	N/R	Yes +IHF	Yes	Acute subdural hematoma with secondary cerebral edema	Yes
4	AI 921001HCC2263	20 mos	F	Platform	1.1/3.5	No	5–10 minutes	Bilateral multilayered	Yes +IHF	Limited	Occipital fracture with subdural/subarachnoid hemorrhage progressing to cerebral edema and herniation	Yes
5[a]	DC 9312060661	23 mos	F	Platform	0.70/2.3	Yes	10 minutes	Bilateral, NOS	Yes	Yes	Acute subdural hematoma	Yes
6	DC 9451016513	26 mos	M	Swing	0.9–1.8/3.0–6.0	Yes	No	Bilateral multilayered	Yes +IHF	Yes	Subdural hematoma with associated cerebral edema	Yes
7[a]	AI 891215HCC2094	3 yrs	M	Platform	0.9/3.0	Yes	10 minutes	N/R	Yes	No	Acute cerebral edema with herniation	No
8	AI 910515HCC2182	3 yrs	F	Ladder	0.6/2.0	yes	15 minutes	N/R	Yes (autopsy only)	Yes	Complex calvarial fracture, contusions, cerebral edema with herniation	Yes
9	DC 9253024577	4 yrs	M	Slide	2.1/7.0	Yes	3 hours	N/R	No	Yes	Epidural hematoma	Yes
10	AI 920710HWE4014	5 yrs	M	Horizontal ladder	2.1/7.0	No	No	N/R	Yes	No	Acute subdural hematoma with acute cerebral edema	Yes
11	AI 960517HCC5175	6 yrs	M	Swing	0.6–2.4/2.0–8.0	No	10 minutes	No	Yes +IHF	No	Acute subdural hematoma	Yes
12	AI 970324HCC3040	6 yrs	M	Horizontal ladder	3.0/10.0	Yes	45 minutes	N/R	No	No	Malignant cerebral edema with herniation	Yes
13	AI 881229HCC3070	6 yrs	F	Horizontal ladder	0.9/3.0	Yes	1+ hour	N/R	Yes +IHF	Yes	Subdural and subarachnoid hemorrhage, cerebral infarct, and edema	Yes
14	AI 930930HWE5025	7 yrs	M	Horizontal ladder	1.2–2.4/4.0–8.0	Yes	48 hours	N/R	No	Yes	Cerebral infarct secondary to carotid/vertebral artery thrombosis	Yes
15	AI 970409HCC1096	8 yrs	F	Retaining wall	0.9/3.0	Yes	12+ hours	N/R	Yes (autopsy only)	Yes	Acute subdural hematoma	Yes
16	AI 890621HCC3195	10 yrs	M	Swing	0.9–1.5/3.0–5.0	Yes	10 minutes	Bilateral multilayered	Yes	Yes	Acute subdural hematoma contiguous with an AV malformation	No
17	AI 920428HCC1671	12 yrs	F	Swing	0.9–1.8/3.0–6.0	Yes	No	N/R	No	Yes	Occipital fracture with extensive contra-coup contusions	Yes
18	AI 891016HCC1511	13 yrs	F	Swing	0.6–1.8/2.0–6.0	Yes	No	N/R	Yes +IHF	Yes	Occipital fracture, subdural hemorrhage, cerebral edema	Yes

[a]The original CT scan for case #7 and the soft tissue CT windows for case #5 could not be located and were unavailable for review.
CPSC, Consumer Products Safety Commission; AI, accident investigation; IR, incident report; DC, death certificate; M, male; F, female; Distance, the distance of the closest body part from the ground at the start of the fall (see text); M/F, meters/feet; Witnessed, witnessed by a noncaretaker or videotaped; N/R, not recorded; IHF, including interhemispheric or falx; FP, forensic pathologist–directed death investigation system.

tubated. A second CT scan demonstrated progression of the left hemispheric edema despite medical management, and he was removed from life support 22 hours after admission.

Case 3

This 17-month-old had been placed in a baby carrier–type swing attached to an overhead tree limb at a daycare provider's home. A restraining bar held in place by a snap was across her waist. She was being pushed by the daycare provider to an estimated height of 1.5 to 1.8 meters (5–6 feet) when the snap came loose. The child fell from the swing on its downstroke, striking her back and head on the grassy surface. She was immediately unconscious and apneic but then started to breathe spontaneously. EMS took her to a pediatric hospital. A CT scan indicated a large left-sided subdural hematoma with extension to the interhemispheric fissure anteriorly and throughout the length of the falx. The hematoma was surgically evacuated, but she developed malignant cerebral edema and died the following day. A postmortem examination indicated symmetrical contusions on the buttock and midline posterior thorax, consistent with impact against a flat surface; a small residual left-sided subdural hematoma; cerebral edema with anoxic encephalopathy; and uncal and cerebellar tonsillar herniation. There were no cortical contusions.

Case 4

A 20-month-old was with other family members for a reunion at a public park. She was on the platform portion of a jungle gym when she fell from the side and struck her head on one of the support posts. The platform was 1.7 meters (67 inches) above the ground and 1.1 meters (42 inches) above the top of the support post that she struck. Only her father saw the actual fall, although there were a number of other people in the immediate area. She was initially conscious and talking, but within 5 to 10 minutes became comatose. She was taken to a nearby hospital, then transferred to a tertiary-care facility. A CT scan indicated a right occipital skull fracture with approximately 4-mm of depression and subarachnoid and subdural hemorrhage along the tentorium and posterior falx. Funduscopic examination indicated extensive bilateral retinal and preretinal hemorrhage. She died 2 days later because of uncontrollable increased intracranial pressure. A limited postmortem examination indicated an impact subgaleal hematoma overlying the fracture in the mid occiput.

Case 5

A 23-month-old was playing on a plastic gym set in the garage at her home with her older brother. She had climbed the attached ladder to the top rail above the platform and was straddling the rail, with her feet 0.70 meters (28 inches) above the floor. She lost her balance and fell headfirst onto a 1-cm (⅜-inch) thick piece of plush carpet remnant covering the concrete floor. She struck the carpet first with her outstretched hands, then with the right front side of her forehead, followed by her right shoulder. Her grandmother had been watching the children play and videotaped the fall. She cried after the fall but was alert and talking. Her grandmother walked/carried her into the kitchen, where her mother gave her a baby analgesic with some water, which she drank. However, approximately 5 minutes later she vomited and became stuporous. EMS personnel airlifted her to a tertiary-care university hospital. A CT scan indicated a large right-sided subdural hematoma with effacement of the right lateral ventricle and minimal subfalcine herniation. (The soft tissue windows for the scan could not be located and were unavailable for review.) The hematoma was immediately evacuated. She remained comatose postoperatively, developed cerebral edema with herniation, and was removed from life support 36 hours after the fall. Bilateral retinal hemorrhage, not further described, was documented in a funduscopic examination performed 24 hours after admission. A postmortem examination confirmed the right frontal scalp impact injury. There was a small residual right subdural hematoma, a right parietal lobe contusion (secondary to the surgical intervention), and cerebral edema with cerebellar tonsillar herniation.

Case 6

A 26-month-old was on a playground swing being pushed by a 13-year-old cousin when he fell backward 0.9 to 1.8 meters (3–6 feet), striking his head on hard-packed soil. The 13-year-old and several other children saw the fall. He was immediately unconscious and was taken to a local emergency room, then transferred to a pediatric hospital. A CT scan indicated acute cerebral edema and a small subdural hematoma adjacent to the anterior interhemispheric falx. A funduscopic examination performed 4 hours after admission indicated extensive bilateral retinal hemorrhage, vitreous hemorrhage in the left eye, and papilledema. He had a subsequent cardiopulmonary arrest and could not be resuscitated. A postmortem examination confirmed the retinal hemorrhage and indicated a right parietal scalp impact injury but no calvarial frac-

ture, a "film" of bilateral subdural hemorrhage, cerebral edema with herniation, and focal hemorrhage in the right posterior midbrain and pons.

Case 7

This 3-year-old with a history of TAR (thrombocytopenia–absent radius) syndrome was playing with other children on playground equipment at his school when he stepped through an opening in a platform. He fell 0.9 meters (3 feet) to the hard-packed ground, striking his face. A teacher witnessed the incident. He was initially conscious and able to walk. However, approximately 10 minutes later he had projectile vomiting and became comatose, was taken to a local hospital, and subsequently transferred to a pediatric hospital. A CT scan indicated a small subdural hematoma and diffuse cerebral edema with uncal herniation, according to the admission history and physical examination. (The original CT report and scan could not be located and were unavailable for review.) His platelet count was 24,000/mm^3, and he was treated empirically with platelet transfusions, although he had no evidence for an expanding extra-axial mass. Resuscitation was discontinued in the emergency room.

Case 8

This 3-year-old was at a city park with an adult neighbor and four other children, ages 6 to 10. She was standing on the third step of a slide ladder 0.6 meters (22 inches) above the ground when she fell forward onto compact dirt, striking her head. The other children but not the adult saw the fall. She was crying but did not appear to be seriously injured, and the neighbor picked her up and brought her to her parents' home. Approximately 15 minutes later she began to vomit, and her mother called EMS. She was taken to a local emergency room, then transferred to a pediatric hospital. She was initially lethargic but responded to hyperventilation and mannitol; she began to open her eyes with stimulation and to spontaneously move all extremities and was extubated. However, she developed malignant cerebral edema on the second hospital day and was reintubated and hyperventilated but died the following day. A postmortem examination indicated a subgaleal hematoma at the vertex of the skull associated with a complex fracture involving the left frontal bone and bilateral temporal bones. There were small epidural and subdural hematomas (not identifiable on the CT scan), bilateral "contra-coup" contusions of the inferior surfaces of the frontal and temporal lobes, and marked cerebral edema with uncal herniation.

Case 9

A 4-year-old fell approximately 2.1 meters (7 feet) from a playground slide at a state park, landing on the dirt ground on his buttock, then falling to his left side, striking his head. There was no loss of consciousness, but his family took him to a local emergency facility, where an evaluation was normal. However, he began vomiting and complained of left neck and head pain approximately 3 hours later. He was taken to a second hospital, where a CT scan indicated a large left parietal epidural hematoma with a midline shift. He was transferred to a pediatric hospital and the hematoma was evacuated, but he developed malignant cerebral edema with right occipital and left parietal infarcts and was removed from the respirator 10 days later. A postmortem examination indicated a small residual epidural hematoma, marked cerebral edema, bilateral cerebellar tonsillar and uncal herniation, and hypoxic encephalopathy. There was no identifiable skull fracture.

Case 10

A 5-year-old was apparently walking across the horizontal ladder of a "monkey bar," part of an interconnecting system of homemade playground equipment in his front yard, when his mother looked out one of the windows and saw him laying face down on the ground and not moving. The horizontal ladder was 2.1 meters (7 feet) above compacted dirt. EMS were called, he was taken to a local hospital, and then transferred to a pediatric hospital. A CT scan indicated a right posterior temporal linear fracture with a small underlying epidural hematoma, a 5-mm thick acute subdural hematoma along the right temporal and parietal lobes, and marked right-sided edema with a 10-mm midline shift. He was hyperventilated and treated with mannitol, but the hematoma continued to enlarge and was surgically evacuated. However, he developed uncontrollable cerebral edema and was removed from life support 10 days after the fall.

Case 11

A 6-year-old was on a playground swing at a private lodge with his 14-year-old sister. His sister heard a "thump," turned around, and saw him on the grass-covered packed earth beneath the swing. The actual fall was not witnessed. The seat of the swing was 0.6 meters (2 feet) above the ground, and the fall distance could have been from as high as 2.4 meters (8 feet). He was initially conscious and talking but within 10 minutes became comatose and was taken to a local emergency room, then transferred to a tertiary-care hospital. A CT

scan indicated a large left frontoparietal subdural hematoma with extension into the anterior interhemispheric fissure and a significant midline shift with obliteration of the left lateral ventricle. There were no retinal hemorrhages. He was treated aggressively with dexamethasone and hyperventilation, but there was no surgical intervention. He died the following day.

Case 12

This 6-year-old was at school and was sitting on the top crossbar of a "monkey bar" approximately 3 meters (10 feet) above compacted clay soil when an unrelated noncaretaker adult saw him fall from the crossbar to the ground. He landed flat on his back and initially appeared to have the wind knocked out of him but was conscious and alert. He was taken to the school nurse who applied an ice pack to a contusion on the back of his head. He rested for approximately 30 minutes in the nurse's office and was being escorted back to class when he suddenly collapsed. EMS was called, and he was transported to a pediatric hospital. He was comatose on admission, the fundi could not be visualized, and a head CT scan was interpreted as normal. However, a CT scan performed the following morning approximately 20 hours after the fall indicated diffuse cerebral edema with effacement of the basilar cisterns and fourth ventricle. There was no identifiable subdural hemorrhage or calvarial fracture. He developed transtentorial herniation and died 48 hours after the fall.

Case 13

This 6-year-old was playing on a school playground with a 5th grade student/friend. She was hand-over-hand traversing the crossbar of a "monkey bar" 2.4 meters (7 feet 10 inches) above the ground with her feet approximately 1 meter (40 inches) above the surface. She attempted to slide down the pole when she reached the end of the crossbar but lost her grip and slid quickly to the ground, striking the compacted dirt first with her feet, then her buttock and back, and finally her head. The friend informed the school principal of the incident, but the child seemed fine and there was no intervention. She went to a relative's home for after-school care approximately 30 minutes after the fall, watched TV for a while, then complained of a headache and laid down for a nap. When her parents arrived at the home later that evening, 6 hours after the incident, they discovered that she was incoherent and "drooling." EMS transported her to a tertiary-care medical center. A CT scan indicated a right parieto-occipital skull fracture, subdural and subarachnoid hemorrhage, and a right cerebral hemisphere infarct. The infarct included the posterior cerebral territory and was thought most consistent with thrombosis or dissection of a right carotid artery that had a persistent fetal origin of the posterior cerebral artery. She remained comatose and was removed from the respirator 6 days after admission. A postmortem examination indicated superficial abrasions and contusions over the scapula, a prominent right parietotemporal subgaleal hematoma, and a right parietal skull fracture. She had a 50-ml subdural hematoma and cerebral edema with global hypoxic or ischemic injury ("respirator brain"), but the carotid vessels were normal.

Case 14

A 7-year-old was on the playground during school hours playing on the horizontal ladder of a "monkey bar" when he slipped and fell 1.2 to 2.4 meters (4–8 feet). According to one witness, he struck his forehead on the bars of the vertical ladder; according to another eyewitness he struck the rubber pad covering of the asphalt ground. There are conflicting stories as to whether he had an initial loss of consciousness. However, he walked back to the school, and EMS was called because of the history of the fall. He was taken to a local hospital, where evaluation indicated a Glasgow coma score of 15 and a normal CT scan except for an occipital subgaleal hematoma. He was kept overnight for observation because of the possible loss of consciousness but was released the following day. He was doing homework at home 2 days after the fall when his grandmother noticed that he was stumbling and had slurred speech, and she took him back to the hospital. A second CT scan indicated a left carotid artery occlusion and left temporal and parietal lobe infarcts. The infarcts and subsequent edema progressed; he had brainstem herniation; and he was removed from life support 3 days later (5 days after the initial fall). A postmortem examination indicated ischemic infarcts of the left parietal, temporal, and occipital lobes, acute cerebral edema with herniation, and thrombosis of the left vertebral artery. Occlusion of the carotid artery, suspected premortem, could not be confirmed.

Case 15

This 8-year-old was at a public playground near her home with several friends her age. She was hanging by her hands from the horizontal ladder of a "monkey bar" with her feet approximately 1.1 meters (3.5 feet) above the ground when she attempted to swing from the bars to a nearby 0.9-

meter (34-inch) retaining wall. She landed on the top of the wall but then lost her balance and fell to the ground, either to a hard-packed surface (one witness) or to a 5.1-cm (2-inch) thick resilient rubber mat (a second witness), striking her back and head. She initially cried and complained of a headache but continued playing, then later went home. Her mother said that she seemed normal and went to bed at her usual time. However, when her mother tried to awaken her at approximately 8:30 the following morning (12 hours after the fall) she complained of a headache and went back to sleep. She awoke at 11 a.m. and complained of a severe headache then became unresponsive and had a seizure. EMS took her to a nearby hospital, but she died in the emergency room. A postmortem examination indicated a right temporoparietal subdural hematoma, extending to the base of the brain in the middle and posterior fossae, with flattening of the gyri and narrowing of the sulci. (The presence or absence of herniation is not described in the autopsy report.) There was no calvarial fracture, and there was no identifiable injury in the scalp or galea.

Case 16

A 10-year-old was swinging on a swing at his school's playground during recess when the seat detached from the chain and he fell 0.9 to 1.5 meters (3–5 feet) to the asphalt surface, striking the back of his head. The other students but not the three adult playground supervisors saw him fall. He remained conscious although groggy and was carried to the school nurse's office, where an ice pack was placed on an occipital contusion. He suddenly lost consciousness approximately 10 minutes later, and EMS took him to a local hospital. He had decerebrate posturing when initially evaluated. Funduscopic examination indicated extensive bilateral confluent and stellate, posterior and peripheral preretinal and subhyaloid hemorrhage. A CT scan showed a large acute right frontoparietal subdural hematoma with transtentorial herniation. The hematoma was surgically removed, but he developed malignant cerebral edema and died 6 days later. A postmortem examination indicated a right parietal subarachnoid AV malformation, contiguous with a small amount of residual subdural hemorrhage, and cerebral edema with anoxic encephalopathy and herniation. There was no calvarial fracture.

Case 17

A 12-year-old was at a public playground with a sister and another friend and was standing on the seat of a swing when the swing began to twist. She lost her balance and fell 0.9 to1.8 meters (3–6 feet) to the asphalt surface, striking her posterior thorax and occipital scalp. She was immediately unconscious and was taken to a tertiary-care hospital emergency room, where she was pronounced dead. A postmortem examination indicated an occipital impact injury associated with an extensive comminuted occipital fracture extending into both middle cranial fossa and "contra-coup" contusions of both inferior frontal and temporal lobes.

Case 18

This 13-year-old was at a public playground with a friend. She was standing on the seat of a swing with her friend seated between her legs when she lost her grip and fell backwards 0.6 to 1.8 meters (2–6 feet), striking either a concrete retaining wall adjacent to the playground or a resilient 5.1-cm (2 inch) thick rubber mat covering the ground. She was immediately unconscious and was given emergency first aid by a physician who was nearby when the fall occurred. She was taken to a nearby hospital and was purposefully moving all extremities and had reactive pupils when initially evaluated. A CT scan indicated interhemispheric subdural hemorrhage and generalized cerebral edema, which progressed rapidly to brain death. A postmortem examination indicated a linear nondepressed midline occipital skull fracture, subdural hemorrhage extending to the occiput, contusion of the left cerebellar hemisphere, bifrontal "contra-coup" contusions, and cerebral edema.

DISCUSSION

General

Traumatic brain injury (TBI) is caused by a force resulting in either strain (deformation/unit length) or stress (force/original cross-sectional area) of the scalp, skull, and brain (35–37). The extent of injury depends not only on the level and duration of force but also on the specific mechanical and geometric properties of the cranial system under loading (38–40). Different parts of the skull and brain have distinct biophysical characteristics, and calculating deformation and stress is complex. However, an applied force causes the skull and brain to move, and acceleration, the time required to reach peak acceleration, and the duration of acceleration may be measured at specific locations (36,41). These kinematic parameters do not cause the actual brain damage but are useful for analyzing TBI because they are easy to quantify. Research in TBI using physical models and animal experiments has shown that a force resulting in angular acceleration pro-

duces primarily diffuse brain damage, whereas a force causing exclusively translational acceleration produces only focal brain damage (36). A fall from a countertop or table is often considered to be exclusively translational and therefore assumed incapable of producing serious injury (3,7–9). However, sudden impact deceleration *must* have an angular vector unless the force is applied only through the center of mass (COM), and deformation of the skull during impact *must* be accompanied by a volume change (cavitation) in the subdural "space" tangential to the applied force (41). The angular and deformation factors produce tensile strains on the surface veins and mechanical distortions of the brain during impact and may cause a subdural hematoma without deep white matter injury or even unconsciousness (42–44).

Many authors state that a fall from less than 3 meters (10 feet) is rarely if ever fatal, especially if the distance is less than 1.5 meters (5 feet) (1–6,8,9). The few studies concluding that a short-distance fall may be fatal (22–24,26,27) have been criticized because the fall was not witnessed or was seen only by the caretaker. However, isolated reports of observed fatal falls and biomechanical analysis using experimental animals, adult human volunteers, and models indicate the potential for serious head injury or death from as little as a 0.6-meter (2-foot) fall (48–52). There are limited experimental studies on infants (cadaver skull fracture) (53,54) and none on living subadult nonhuman primates, but the adult data have been extrapolated to youngsters and used to develop the Hybrid II/III and Child Restraint–Air Bag Interaction (CRABI) models (55) and to propose standards for playground equipment (56,63). We simply do not know either kinematic or nonkinematic limits in the pediatric population (57,58).

Each of the falls in this study exceeded established adult kinematic thresholds for traumatic brain injury (41,48–52). Casual analysis of the falls suggests that most were primarily translational. However, deformation and *internal* angular acceleration of the skull and brain *caused by the impact* produce the injury. What happens during the impact, not during the fall, determines the outcome.

Subdural Hemorrhage

A "high strain" impact (short pulse duration and high rate for deceleration onset) typical for a fall is more likely to cause subdural hemorrhage than a "low strain" impact (long pulse duration and low rate for deceleration onset) that is typical of a motor vehicle accident (42,61). The duration of deceleration for a head-impact fall against a nonyielding surface is usually less than 5 milliseconds (39,59–61). Experimentally, impact duration longer than 5 milliseconds will not cause a subdural hematoma unless the level of angular acceleration is above 1.75×10^5 rad/s^2 (61). A body in motion with an angular acceleration of 1.75×10^5 rad/s^2 has a tangential acceleration of 17,500 m/s^2 at 0.1 meters (the distance from the midneck axis of rotation to the midbrain COM in the Duhaime model). A human cannot produce this level of acceleration by impulse ("shake") loading (62).

An injury resulting in a subdural hematoma in an infant may be caused by an accidental fall (43,44,64). A recent report documented the findings in seven children seen in a pediatric hospital emergency room after an accidental fall of 0.6 to 1.5 meters who had subdural hemorrhage, no loss of consciousness, and no symptoms (44). The characteristics of the hemorrhage, especially extension into the posterior interhemispheric fissure, have been used to suggest if not confirm that the injury was nonaccidental (9,62,65–68). The hemorrhage extended into the posterior interhemispheric fissure in 5 of the 10 children in this study (in whom the blood was identifiable on CT or magnetic resonance scans and the scans were available for review) and along the anterior falx or anterior interhemispheric fissure in an additional 2 of the 10.

Lucid Interval

Disruption of the diencephalic and midbrain portions of the reticular activating system (RAS) causes unconsciousness (36,69,70). "Shearing" or "diffuse axonal" injury (DAI) is thought to be the primary biophysical mechanism for immediate traumatic unconsciousness (36,71). Axonal injury has been confirmed at autopsy in persons who had a brief loss of consciousness after a head injury and who later died from other causes, such as coronary artery disease (72). However, if unconsciousness is momentary or brief ("concussion") subsequent deterioration *must* be due to a mechanism other than DAI. Apnea and catecholamine release have been suggested as significant factors in the outcome following head injury (73,74). In addition, the centripetal theory of traumatic unconsciousness states that primary disruption of the RAS will not occur in isolation and that structural brainstem damage from inertial (impulse) or impact (contact) loading *must* be accompanied by evidence for cortical and subcortical damage (36). This theory has been validated by magnetic resonance imaging and CT scans in adults and children (75,76). Only one of the children in this study (case 6) had evidence for any component of DAI. This child had focal hemor-

rhage in the posterior midbrain and pons, thought by the pathologist to be primary, although there was no skull fracture, only "a film" of subdural hemorrhage, no tears in the corpus callosum, and no lacerations of the cerebral white matter (grossly or microscopically).

The usual cause for delayed deterioration in infants and children is cerebral edema, whereas in adults it is an expanding extra-axial hematoma (77). If the mechanism for delayed deterioration (except for an expanding extra-axial mass) is venospasm, cerebral edema may be the only morphologic marker. The "talk and die or deteriorate (TADD)" syndrome is well characterized in adults (78). Two reports in the pediatric literature discuss TADD, documenting 4 fatalities among 105 children who had a lucid interval after head injury and subsequently deteriorated (77,79). Many physicians believe that a lucid interval in an ultimately fatal pediatric head injury is extremely unlikely or does not occur unless there is an epidural hematoma (7,8,11). Twelve children in this study had a lucid interval. A noncaretaker witnessed 9 of these 12 falls. One child had an epidural hematoma.

Retinal Hemorrhage

The majority of published studies conclude that retinal hemorrhage, especially if bilateral and posterior or associated with retinoschisis, is highly suggestive of, if not diagnostic for, nonaccidental injury (9,14–21). Rarely, retinal hemorrhage has been associated with an accidental head injury, but in these cases the bleeding was unilateral (80). It is also stated that traumatic retinal hemorrhage may be the direct mechanical effect of violent shaking (15). However, retinal hemorrhage may be caused experimentally either by ligating the central retinal vein or its tributaries or by suddenly increasing intracranial pressure (81,82); retinoschisis is the result of breakthrough bleeding and venous stasis not "violent shaking" (15,83). Any sudden increase in intracranial pressure may cause retinal hemorrhage (84–87). Deformation of the skull coincident to an impact nonselectively increases intracranial pressure. Venospasm secondary to traumatic brain injury selectively increases venous pressure. Either mechanism may cause retinal hemorrhage irrespective of whether the trauma was accidental or inflicted. Further, retinal and optic nerve sheath hemorrhages associated with a ruptured vascular malformation are due to an increase in venous pressure not extension of blood along extravascular spaces (81–83,88). Dilated eye examination with an indirect ophthalmoscope is thought to be more sensitive for detecting retinal bleeding than routine examination and has been recommended as part of the evaluation of any pediatric patient with head trauma (89). None of the children in this study had a formal retinal evaluation, and only six had funduscopic examination documented in the medical record. Four of the six had bilateral retinal hemorrhage.

Pre-existing Conditions

One of these children (case 16) had a subarachnoid AV malformation that contributed to development of the subdural hematoma, causing his death. One (case 7) had TAR syndrome (90), but his death was thought to be caused by malignant cerebral edema not an expanding extra-axial mass.

Cerebrovascular Thrombosis

Thrombosis or dissection of carotid or vertebral arteries as a cause of delayed deterioration after head or neck injuries is documented in both adults and children (91,92). Case 14 is the first report of a death due to traumatic cerebrovascular thrombosis in an infant or child. Internal carotid artery thrombosis was suggested radiographically in an additional death (case 13) but could not be confirmed at autopsy. However, this child died 6 days after admission to the hospital, and fibrinolysis may have removed any evidence for thrombosis at the time the autopsy was performed.

Limitations

1. Six of the 18 falls were not witnessed or were seen only by the adult caretaker, and it is possible that another person caused the nonobserved injuries.
2. The exact height of the fall could be determined in only 10 cases. The others (7 swing and 1 stationary platform) could have been from as little as 0.6 meters (2 feet) to as much as 2.4 meters (8 feet).
3. A minimum impact velocity sufficient to cause fatal brain injury cannot be inferred from this study. Likewise, the probability that an individual fall will have a fatal outcome cannot be stated because the database depends on voluntary reporting and contractual agreements with selected U.S. state agencies. The NEISS summaries for the study years estimated that there were more than 250 deaths due to head and neck injuries associated with playground equipment, but there are only 114 in the files. Further, this study does not include other nonplayground equipment–related fatal falls, witnessed or not witnessed, in the CPSC database (32).

CONCLUSIONS

1. Every fall is a complex event. There must be a biomechanical analysis for any incident in which the severity of the injury appears to be inconsistent with the history. The question is not "Can an infant or child be seriously injured or killed from a short-distance fall?" but rather "If a child falls (x) meters and strikes his or her head on a nonyielding surface, what will happen?"
2. Retinal hemorrhage may occur whenever intracranial pressure exceeds venous pressure or whenever there is venous obstruction. The characteristic of the bleeding cannot be used to determine the ultimate cause.
3. Axonal damage is unlikely to be the mechanism for lethal injury in a low-velocity impact such as from a fall.
4. Cerebrovascular thrombosis or dissection must be considered in any injury with apparent delayed deterioration, and especially in one with a cerebral infarct or an unusual distribution for cerebral edema.
5. A fall from less than 3 meters (10 feet) in an infant or child may cause fatal head injury and may not cause immediate symptoms. The injury may be associated with bilateral retinal hemorrhage, and an associated subdural hematoma may extend into the interhemispheric fissure. A history by the caretaker that the child may have fallen cannot be dismissed.

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APPENDIX

Newtonian mechanics involving constant acceleration may be used to determine the impact velocity in a gravitational fall. However, constant acceleration formulas cannot be used to calculate the relations among velocity, acceleration, and distance traveled *during* an impact because the deceleration is not uniform (45). This analysis requires awareness of the shape of the deceleration curve, knowledge of the mechanical properties and geometry of the cranial system, and comprehension of the stress and strain characteristics for the specific part of the skull and brain that strikes the ground. A purely translational fall requires that the body is rigid and that the external forces acting on the body pass only through the COM, i.e., there is no rotational component. A 1-meter-tall 3-year-old hanging by her knees from a horizontal ladder with the vertex of her skull 0.5 meters above hard-packed earth approximates this model. If she looses her grip and falls, striking the occipital scalp, her impact velocity is 3.1 m/second. An exclusively angular fall also requires that the body is rigid. In addition, the rotation must be about a fixed axis or a given point internal or external to the body, and the applied moment and the inertial moment must be at the identical point or axis. If this same child has a 0.5-meter COM and has a "matchstick" fall while standing on the ground, again striking her occiput, her angular velocity is 5.42 rad/second and tangential velocity 5.42 m/second at impact. The impact velocity is higher than predicted for an exclusively translational or external-axis angular fall when the applied moment and the inertial moment are at a different fixed point (slip and fall) or when the initial velocity is not zero (walking or running, then trip and fall), and the vectors are additive. However, the head, neck, limbs, and torso do not move uniformly during a fall because relative motion occurs with different velocities and accelerations for each component. Calculation of the impact velocity for an actual fall requires solutions of differential equations for each simultaneous translational and rotational motion (45). Further, inertial or impulse loading (whiplash) may cause head acceleration more than twice that of the midbody input force and may be important in a fall where the initial impact is to the feet, buttock, back, or shoulder, and the final impact is to the head (46,47).

The translational motion of a rigid body at constant gravitational acceleration (9.8 m/s^2) is calculated from:

$$F = ma \qquad v^2 = 2as \qquad v = at$$

where F = the sum of all forces acting on the body (newton), m = mass (kg), a = acceleration (m/s^2), v = velocity (m/s), s = distance (m), and t = time (s).

The angular motion of a rigid body about a fixed axis at a given point of the body under constant gravitational acceleration (9.8 m/s^2) is calculated from:

$$M = I\alpha \qquad \omega = v^1/r \qquad \alpha = a^1/r$$

where M = the applied moment about the COM or about the fixed point where the axis of rotation is located, I = the inertial moment about this same COM or fixed point, α = angular acceleration (rad/s^2), ω = angular velocity (rad/s), r = radius (m), v^t = tangential velocity (m/s), and a^t = tangential acceleration (m/s^2).

The angular velocity ω for a rigid body of length L rotating about a fixed point is calculated from:

$$\tfrac{1}{2}I_0\omega^2 = maL/2 \qquad I_0 = (1/3)\,mL^2$$

where I_0 = the initial inertial moment, ω = angular velocity (rad/s), m = mass (kg), a = gravitational acceleration (9.8 m/s^2), and L = length.

REFERENCES

1. Chadwick DL, Chin S, Salerno C, et al. Deaths from falls in children: how far is fatal? *J Trauma* 1991;31:1353–5.
2. Williams RA. Injuries in infants and small children resulting from witnessed and corroborated free falls. *J Trauma* 1991;31:1350–2.
3. Duhaime AC, Alario AJ, Lewander WJ, et al. Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 1992;90:179–85.
4. Lyons TJ, Oates RK. Falling out of bed: a relatively benign occurrence. *Pediatrics* 1993;92:125–7.
5. Swalwell C. Head injuries form short distance falls. *Am J Forensic Med Pathol* 1993;14:171–2.
6. Sheridan F, Anthony RM, Rieber GD, et al. Head injuries from short distance falls. *Am J Forensic Med Pathol* 1993; 14:172–3.
7. Duhaime AC, Christian CW, Rorke LB, et al. Non-accidental head injury in infants: the "shaken baby syndrome." *N Engl J Med* 1998;338:1822–1829.
8. Case ME, Graham MA, Corey Handy T, et al. Position paper on shaken baby. Adopted by the Board of Directors, United States National Association of Medical Examiners, San Francisco, October 30, 1998. St. Louis: The National Association of Medical Examiners.
9. Showers J. Never shake a baby. The Second National Conference on Shaken Baby Syndrome. National Association of Children's Hospitals and Related Institutions, 1999. Available at: www.childrenshospitals.net/nachri/news/pr%5Fsbs.html.
10. Tullous M, Walker MD, Wright LC. Evaluation and treatment of head injuries in children. In Fuhrman BP, Zimmerman JJ, eds. *Pediatric critical care*. St. Louis: Mosby Year Book, 1992:1165–82.
11. Willman KY, Bank DE, Senac M, Chadwick DL. Restricting the time of injury in fatal inflicted head injury. *Child Abuse Negl* 1997;21:929–40.
12. Amaya M, Bechtel K, Blatt SD, et al. Shaken baby syndrome and the death of Matthew Eappen. (November 11, 1997). Available at: www.silcon.com/∼ptave/shaken.htm.
13. Jenny C, Hymel KP. Recognizing abusive head trauma in children. *JAMA* 1999;282:1421–2.
14. Eisenbrey AB. Retinal hemorrhage in the battered child. *Childs Brain* 1979;5:40–4.
15. Greenwald MJ, Weiss A, Oesterle CS, et al. Traumatic retinoschisis in battered babies. *Ophthalmology* 1986;93: 618–24.
16. Rao N, Smith RE, Choi JH, et al. Autopsy findings in the eyes of fourteen fatally abused children. *Forensic Sci Int* 1988;39:293–9.
17. Elner SG, Elner VM, Arnall M, Albert DM. Ocular and associated systemic findings in suspected child abuse: a necropsy study. *Arch Opthalmol* 1990;108:1094–101.
18. Williams DF, Swengel RM, Scharre DW. Posterior segment manifestations of ocular trauma. *Retina* 1990;10 (suppl):535–44.
19. Rosenberg NM, Singer J, Bolte R, et al. Retinal hemorrhage. *Pediatr Emerg Care* 1994;10:303–5.
20. Swenson J, Levitt C. Shaken baby syndrome: diagnosis and prevention. *Minn Med* 1997;80:41–4.
21. Altman RL, Kutscher ML, Brand DA. The "shaken-baby syndrome." *N Engl J Med* 1998;339:1329–30.
22. Hall JR, Reyes HM, Horvat M, et al. The mortality of childhood falls. *J Trauma* 1989;29:1273–5.
23. Rieber GD. Fatal falls in childhood: how far must children fall to sustain fatal head injury: report of cases and review of the literature. *Am J Forensic Med Pathol* 1993;14: 201–7.
24. Root I. Head injuries from short distance falls. *Am J Forensic Med Pathol* 1992;13:85–7.
25. Nashelsky MB, Dix JD. The time interval between lethal infant shaking and onset of symptoms: a review of the shaken baby syndrome literature. *Am J Forensic Med Pathol* 1995;16:154–7.
26. Wilkins B. Head injury: abuse or accident? *Arch Dis Child* 1997;76:393–7.
27. Shaken babies (editorial). *Lancet* 1998;352:335.
28. Plunkett J. Restricting the time of injury in fatal inflicted head injuries. *Child Abuse Negl* 1998;22:943–4.
29. Sweeney TB. X-rated playgrounds? *Pediatrics* 1979;64: 961.
30. Tursz A, LeLong N, Crost M. Home accidents to children under 2 years of age. *Paediatr Perinatol Epidemiol* 1990; 4:408–21.
31. National Injury Information Clearinghouse. US Consumer Product Safety Commission (1997). Washington DC 20207. Available at: www.cpsc.gov/about/clrnghse.html.
32. Consumer Product Safety Review 1999;4:#2:3–7. Available at: www.cpsc.gov/cpscpub/pubs/cpsr.html.
33. A description of the injury or potential injury incident data base (IPII). Division of Hazard and Injury Data Systems. US Consumer Product Safety Commission (1997). Washington DC 20207. Available at: www.cpsc.gov/about/guide.html#OIPA.
34. A description of the indepth investigation database (INDP), fiscal year 1987–fiscal year 1991. Division of Hazard and Injury Data Systems. US Consumer Product Safety Commission (1992). Washington D.C. 20207. Available at: www.cpsc.gov/about/guide.html#OIPA.
35. Ommaya AK, Gennarelli TA. Cerebral concussion and traumatic unconsciousness. *Brain* 1974;97:633–54.
36. Ommaya AK. Head injury mechanisms and the concept of preventive management: a review and critical synthesis. *J Neurotrauma* 1995;12:527–46.
37. Gurdjian ES, Hodgson VR, Thomas LM, Patrick LM. Significance of relative movements of scalp, skull and intracranial contents during impact injury of the head. *J Neurosurg* 1968;29:70–2.
38. Ommaya AK, Grubb RL, Naumann RA. Coup and contra-coup injury: observations on the mechanics of visible brain injuries in the rhesus monkey. *J Neurosurg* 1971;35: 503–16.
39. Gurdjian ES. Recent advances in the study of the mechanism of impact injury of the head: a summary. *Clin Neurosurg* 1972;19:1–42.
40. Gennarelli TA, Thibault LE, Adams JH, et al. Diffuse axonal injury and traumatic coma in the primate. *Ann Neurol* 1982;12:564–74.
41. McElhaney JH, Roberts VL, Hilyard JF. Head injury tolerance and criteria. In *Handbook of human tolerance*. Yatabecho, Tukuba-gun, Ibaraki, Japan: Japan Automobile Research Institute, Inc., 1976:237–335.
42. Gurdjian ES, Hodgson VR, Thomas LM, Patrick LM. Impact head injury: mechanism and prevention. In Brinkhous

KM (ed), *Accident pathology.* Proceedings of an International Conference; June 6–8, 1968; Washington DC (US). USDT, FHA, NHSB #FH 11-6595, 1968;140–143.
43. Aoki N, Masuzawa H. Infantile acute subdural hematoma. *J Neurosurg* 1984;61:272–80.
44. Greenes DS, Schutzman SA. Occult intracranial injury in infants. *Ann Emerg Med* 1998;32:680–6.
45. Berger SA, Goldsmith W, Lewis ER, eds. *Introduction to bioengineering.* New York: Oxford University Press, 1996.
46. Ewing, CL, Thomas DJ, Patrick LM, et al. Living human dynamic response to Gx impact acceleration. II. Accelerations measured on the head and neck. Proceedings, Thirteenth Stapp Car Crash Conference, 1969, December 2–4, Boston, MA. New York: Society of Automotive Engineers, Inc., 1969:400–15.
47. Ommaya AK, Yarnell P. Subdural haematoma after whiplash injury. *Lancet* 1969;294:237–9.
48. Gurdjian ES, Lissner HR, Patrick LM. Protection of the head and neck in sports. *JAMA* 1962;182:509–12.
49. Gurdjian ES, Roberts UL, Thomas LM. Tolerance curves of acceleration and intracranial pressure and protective index in experimental head injury. *J Trauma* 1966;6:600–4.
50. Mahajan BM, Beine WB. Impact attenuation performance of surfaces installed under playground equipment: report to the Consumer Product Safety Commission. US Department of Commerce, National Bureau of Standards, Washington, DC: Feb 1979. Publication No. NBSIR 79-1707.
51. Reichelderfer TE, Overbach A, Greensher J. X-rated playgrounds? *Pediatrics* 1979;64:962–3.
52. Collantes M. Playground surfaces and head injury: evaluation of the importance of using the Head Injury Criterion (HIC) to estimate the likelihood of head impact injury as a result of a fall onto playground surface materials. US Consumer Product Safety Commission (1990). Washington D.C. 20207. Available at: www.cpsc.gov/cpscpub/pubs/3005.html.
53. Weber W. Experimental studies of skull fractures in infants. *Z Rechtsmed* 1984;92:87–94.
54. Weber W. Biomechanical fragility of the infant skull. *Z Rechtsmed* 1985;94:93–101.
55. Irwin A, Mertz HJ. Biomechanical basis for the CRABI and Hybrid III child dummies. New York: Society of Automotive Engineers, Inc., Document #973317, 1997:261–72.
56. *Handbook for public playground safety.* Washington, DC: US Consumer Product Safety Commission, Pub No. 325, 1997. Available at: www.cpsc.gov/cpscpub/325.html.
57. Goldsmith W. Current controversies in the stipulation of head injury criteria. *J Biomech* 1981;12:883–4.
58. Goldsmith W. Meaningful concepts of head injury criteria. In Proceedings of IRCOBI Conference; 1989 Sept 13–15; Stockholm, Sweden. France: Bron, IRCOBI Secretariat; 1989:1–11.
59. Gurdjian ES. The mechanism of skull fracture. *J Neurosurg* 1950;7:106–14.
60. Evans FG, Lissner HR, Lebow M. The relation of energy, velocity, and acceleration to skull deformation and fracture. *Surg Gynicol Obstet* 1958;106:593–601.
61. Gennarelli TA, Thibault LE. Biomechanics of acute subdural hematoma. *J Trauma* 1982;22:680–6.
62. Duhaime AC, Gennarelli TA, Thibault LE, et al. The shaken baby syndrome: a clinical, pathological and biomechanical study. *J Neurosurg* 1987;66:409–15.
63. Cory CZ. Assessing the severity of child head impacts onto various domestic surface mixtures. MPhil Thesis, School of Engineering, Cardiff University, Wales (UK). May 1998. Available at: http://library.cf.ac.uk/
64. Howard MA, Bell BA, Uttley D. The pathophysiology of infant subdural haematomas. *Br J Neurosurg* 1993;7:355–65.
65. Zimmerman RA, Bilaniuk LT, Bruce D, et al. Computed tomography of craniocerebral injury in the abused child. *Radiology* 1979;130:687–90.
66. Merten DF, Osborne DRS. Craniocerebral trauma in the child abuse syndrome. *Pediatr Ann* 1983;12:882–7.
67. Merten DF, Osborne DRS, Radkowski MA, Leonidas JC. Craniocerebral trauma in the child abuse syndrome: radiologic observations. *Pediatr Radiol* 1984;14:272–7.
68. Kleinman PD. Head trauma. In Kleinman PK (ed). *Diagnostic imaging of child abuse.* Baltimore: Williams & Wilkins, 1987:159–99.
69. Magoun HW. *The waking brain.* Springfield, IL: Thomas, 1958.
70. Plum F, Posner JB. *The diagnosis of stupor and coma.* Philadelphia: Davis; 1966.
71. Blumbergs PC, Jones NR, North JB. Diffuse axonal injury in head trauma. *J Neurol Neurosurg Psychiatry* 1989;52:838–41.
72. Oppenheimer DR. Microscopic lesions in the brain following head injury. *J Neurol Neurosurg Psychiatry* 1968;31:299–306.
73. Johnson DL, Boal D, Baule R. Role of apnea in non-accidental head injury. *Pediatr Neurosurg* 1995;23:305–10.
74. Atkinson JLD. The neglected pre-hospital phase of head injury: apnea and catecholamine surge. *Mayo Clin Proc* 2000;75:37–47.
75. Teasdale E, Cardoso E, Galbraith S, Teasdale G. CT scans in severe diffuse head injury: physiopathology and clinical correlations. *J Neurol Neurosurg Psychiatry* 1984;47:600–3.
76. Levin HS, Mendelsohn D, Lilly MA, et al. Magnetic resonance imaging in relation to functional outcome of pediatric closed head injury: a test of the Ommaya-Gennarelli model. *Neurosurgery* 1997;40:432–41.
77. Bruce DA, Alavi A, Bilaniuk L, et al. Diffuse cerebral swelling following head injuries in children: the syndrome of "malignant brain edema." *J Neurosurg* 1981;54:170–8.
78. Rockswold GL, Leonard RP, Nagib MG. Analysis of management in thirty-three closed head injury patients who "talked and deteriorated." *Neurosurgery* 1987;21:51–5.
79. Snoek JW, Minderhoud JM, Wilmink JT. Delayed deterioration following mild head injury in children. *Brain* 1984;107:15–36.
80. Christian CW, Taylor AA, Hertle RW, Duhaime AC. Retinal hemorrhage caused by accidental household trauma. *J Pediatr* 1999;135:125–7.
81. Smith DC, Kearns TP, Sayre GP. Pre-retinal and optic nerve sheath hemorrhage: pathologic and experimental aspects in subarachnoid hemorrhage. *Trans Am Acad Ophthalmol Otolaryngol* 1957;61:201–11.
82. Lehman RAW, Krupin T, Podos SM. Experimental effect of intracranial hypertension upon intraocular pressure. *J Neurosurg* 1972;36:60–6.
83. Vanderlinden RG, Chisholm LD. Vitreous hemorrhages and sudden increased intracranial pressure. *J Neurosurg* 1974;41:167–76.
84. Kirschner RH, Stein RJ. The mistaken diagnosis of child abuse: a form of medical abuse? *Am J Dis Child* 1985;139:873–5.
85. Weedn VW, Mansour AM, Nichols MM. Retinal hemorrhage in an infant after cardiopulmonary resuscitation. *Am J Forensic Med Pathol* 1990;11:79–82.
86. David DB, Mears T, Quinlan MP. Ocular complications associated with bungee jumping. *Br J Ophthalmol* 1994;78:234–5.
87. Jain BK, Talbot EM. Bungee jumping and intraocular hemorrhage. *Br J Ophthalmol* 1994;78:236–7.
88. Edlow JA, Caplan LR. Avoiding pitfalls in the diagnosis of subarachnoid hemorrhage. *N Engl J Med* 2000;342:29–36.
89. Becker H, Gupta BK. Recognizing abusive head trauma in children. *JAMA* 1999;282:1421.
90. Hall JG, Levin J, Kuhn JP, et al. Thrombocytopenia with absent radius (TAR). *Medicine (Baltimore)* 1969;48:411–39.
91. Pozzati E, Giuliani G, Poppi M, Faenza A. Blunt traumatic carotid dissection with delayed symptoms. *Stroke* 1989;20:412–6.
92. Martin PJ, Enevoldson TP, Humphrey PRD. Causes of ischaemic stroke in the young. *Postgrad Med J* 1997;73:8–16.

Addendum

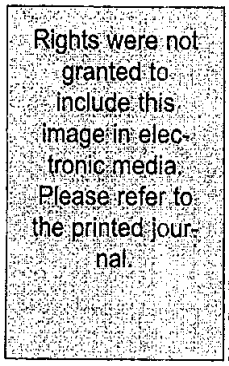
This death was reported to and investigated by the CPSC after submission of the manuscript.

Case 19. A 7 year old boy was playing at a school playground, hanging by his hands from the gymnastic rings of a wooden play structure with his feet approximately 30 inches above the ground. Another child grabbed him by the legs, forcing him to let go, and he fell face-first onto 7 inches of wood mulch, which had been placed on the playground surface the day before. The fall was witnessed by several other children and adults. He seemed uninjured and continued playing. However, 10-15 minutes later he complained of a headache and went to the school nurse's office to lay down. He had a seizure and lost consciousness while in the office, was transported to a local hospital, then transferred to a tertiary care children's hospital. A CT scan indicated acute occipito-parietal subdural hemorrhage, extending along the tentorium and posterior interhemispheric fissure. The hematoma was emergently evacuated, but he had a cardiopulmonary arrest in the operating room and could not be resuscitated. A postmortem examination indicated residual subdural hemorrhage at the base of the skull. He had no impact injury in his scalp, consistent with the history of a face-first fall, and had no identifiable facial lacerations or abrasions. The neck and cervical spinal cord examination were normal. The eyes were not examined.

John Plunkett, M.D.
September 12, 2000

Is gene therapy ready for HIV/Ebola virus-derived viral vectors?

In 1999, an investigation into the death of Jesse Gelsinger, who died while particpating in a gene theapy trial, severely criticised James Wilson, director of the Institute of Gene Therapy at the University of Pennsylvania (Philadelphia, PA, USA). The controversy is revisited this month with the publication of a paper by Wilson's laboratory reporting the development of an HIV-based viral vector that carries envelope proteins from the Ebola virus.



Science Photo Library

Using Ebola envelope proteins

The group suggest that the new vector, EboZ, which efficiently transduces intact airway epithelium in vitro and in vivo, may form the basis of an effective gene therapy for cystic fibrosis. "At a time when gene therapists, the FDA, and many others in the field are struggling for restoration of public confidence in gene therapy, one might question the approach of creating a 'strange bug' instead of optimising the known viral or even nonviral transfer technologies", says Wolfgang Walther (Max-Delbrück-Center for Molecular Medicine, Berlin, Germany).

Wilson's team created vectors that incorporated various viral envelope proteins and showed that a vector containing envelope proteins from the Zaire strain of Ebola virus was the most effective transducer of cultured apical airway cells in culture. Further in-vitro experiments on excised sections of healthy human trachea demonstrated transduction of tracheal epithelial cells by the EboZ vector. This was followed up by in-vivo experiments in which the vector was introduced into the tracheas of immunocompetent young mice—the animals had high-levels of vector expression by day 28, that persisted until at least day 63 (*Nat Biotech* 2001; 19: 225–30).

"The EboZ vector construct serves as a research tool and provides the means to ask if there is a single epitope in the Ebola virus envelope that is critical for binding the receptors on a respiratory epithelial cell", stresses Nelson A Wivel, deputy director of the Institute of Gene Therapy in Philadelphia. "The idea of employing Ebola envelopes to achieve transduction of airway epithelium—the natural target of Ebola infection—is intriguing", agrees Walther, but he warns that safety concerns are under-represented in the study. "At least one experiment should have investigated whether cell types other than epithelial cells can be infected by the new vector", he says. A scenario of efficient but unwanted infection of other tissues could rule out use of the vector for human gene therapy, he adds.

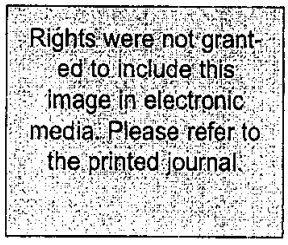
Gaetano Romano (Thomas Jefferson University, Philadelphia, PA, USA) also warns that a major drawback of HIV-based vectors is the seroconversion to HIV. He also points out that insertion of the viral vector into the genome of human cells and possible recombination between retroviridae-based vectors and human endogenous retroviruses need to be considered. Insertional mutagenesis could be avoided by engineering self-inactivating vectors but Romano notes that "the transfer vector used by Wilson belongs to the early generations of HIV-based vectors, which are not self-inactivating." "Obviously, many more studies need to be done and the question of a clinical trial is very remote in our thinking at this juncture", says Wivel.

Kathryn Senior

Accident or murder in children?

In 1998, there were several well-publicised trials of child carers who were accused of killing children in their care by shaking them. Experts for the prosecution gave evidence that there were features of the fatal event, and physical signs in the children, that were diagnostic of inflicted injury, although the evidence underlying their assertions was slight. Many observers, including *The Lancet* (1998; 352: 335), expressed concern at this deficiency.

John Plunkett from the Regina Medical Center, Hastings MN, USA, examined the records of the United States Consumer Products Safety Commission between January, 1988, and June, 1999, to find the records of children who died after short falls (0·6–3 m) from playground equipment (*Am J Forensic Med Pathol* 2001; 22: 1–12). 18 children were identified, aged 12 months to 13 years. Legal investigations concluded that death was accidental in all cases. A non-caretaker witnessed 12 of the accidents, and in the 13th the fall was videotaped by the child's grandmother.



Kevin Harrison/Medipics

Is retinal haemorrhage diagnostic?

"Many physicians believe that . . . a lucid interval does not exist in an ultimately fatal paediatric head injury", says Plunkett, yet 12 of the 18 children who died had a lucid interval lasting from 5 min to 48 h. Four of the six children whose fundi were examined had bilateral retinal haemorrhages, which contradicts the assumption, "that retinal haemorrhage is highly suggestive, if not diagnostic, of inflicted trauma".

The author's conclusion that, "a history by the caretaker that the child may have fallen cannot be dismissed", is likely to echo through courtrooms for many years to come. He is more forthright in person about the issue: "I am genuinely distressed at what medicine has done in the arena of child abuse. Even a cursory understanding of the biomechanics of brain trauma would have predicted the results I was fortunately able to document . . . It [the publication] has already been 'trashed' by many paediatricians and ophthalmologists, and the journal was not mailed until last Friday [Feb 23]! It has even been suggested that the videotape of the fatal short-distance fall was fabricated."

John Bignall

The American Journal of Forensic Medicine and Pathology 22(3):332-336, 2001.

Letters to the Editor

Fatal Pediatric Head Injuries Caused by Short-Distance Falls

To the Editor:

The case series drawn from Consumer Product Safety Commission (CPSC) databases (1) is the largest series of reasonably well-described deaths in childhood resulting from falls of less than 10 feet. Only a few other cases have been this well reported (2,3). For this reason, therefore, it is unfortunate that not one computed tomography scan or autopsy photograph was included to amplify or elucidate any of the reported findings. Similarly, the lack of detailed neuropathologic descriptions limits the reader's ability to define mechanism of injury. For example, four of the seven cases with skull fracture had fractures involving the occipital bone (cases 4, 13, 17, and 18). In three of these four (cases 4, 13, and 18), acute subdural hematoma (ASDH) was part of the reported findings. Fractures in the occipital bone have the potential for laceration of the sagittal or transverse sinus. This would cause a large mass-effect, subdural hematoma arising as a contact injury. This should be contrasted with ASDH, which results from the rupture of bridging veins and which may happen focally due to contact forces or more widely due to inertial forces.

The series establishes the extreme rarity of death from head injury in young children who fall short distances. The CPSC reports that over 200,000 children are seen in emergency departments each year because of injuries incurred while using playground equipment. Sixty percent (approximately 120,000) of these are the result of falls (4). Over the 11.5-year period covered in Plunkett's report, more than 1.3 million such emergency department visits would be expected. However, only 18 deaths from head injury were identified. This yields a rate of 1.3 deaths per 100,000 such falls. In fact, since we may presume that most children who fall from playground equipment are not brought to an emergency department, the fatality rate must be considerably lower than this. By anyone's standards, such deaths are very rare events. Included in this small series are two cases (cases 7 and 16) in which natural causes (thrombocytopenia–absent radii syndrome, intracranial A-V malformation) may have increased susceptibility to fatal head injury. If these two cases are excluded, the fatality rate drops to 1.16 per 100,000 children brought to an emergency department after a playground equipment fall.

The series also establishes that skull fracture is much less commonly associated with fatal head injury in children than in adults. Eleven (61%) of the 18 children described by Plunkett had no skull fracture (cases 2, 3, 5–7, 9, 11, 12, 14–16), despite falls of 0.6 to 2.4 meters (approximately 2–8 feet). This finding is in agreement with the few reports of fatal falls from short distances that include enough pathologic description to ensure their reasonable validity. In the three cases described by Claydon (2) and Reiber (3), none of the children had skull fractures.

The findings in this series once again cast serious doubt on the validity of the two papers by Weber (5,6) cited in this report. If Weber's conclusions were valid, all of these children should have had skull fractures. A recent paper (7) establishes the ability of the infant skull to deform without fracture and, in fact, indicates that such transient deformation may actually contribute to the severity of a head injury associated with impact.

The article states that all of the 18 cases reported here reached expected biomechanical thresholds for traumatic brain injury in adults, but no such analysis is provided in even one case. Without such a description, there is no way to evaluate the validity of the analysis.

It is extremely interesting that 6 of the 18 cases (cases 1, 3, 6, 16–18) involved falls from swings. It is unfortunate that Plunkett did not take the opportunity to discuss the physics of swing injuries. With regard to linear deceleration at impact, the distance from the ground at the moment the child falls from the swing is inconsequential. The determining factor in the terminal velocity prior to impact will be the maximum height attained during the swing cycle. As the swing descends through the arc, increasing amounts of the potential energy are transformed into kinetic energy and therefore into linear velocity. Thus, the energy available at impact, and

hence the terminal velocity, is equivalent to a fall from the maximum height of the arc. In fact, the nadir of the swing cycle is associated with the highest angular velocity. When a child falls from a swing, he begins to rotate around his own center of gravity, rather than around the support beam, which may be as much as 8 feet from the swing seat. This leads to an immediate and marked decrease in the moment of inertia. Preservation of angular momentum, then, will cause the child's angular velocity to increase. If the child has fallen from the swing at a point low in the descending arc, when angular velocity is higher to begin with, this further increase in angular velocity may lead to very high angular decelerations at impact. Since angular acceleration and velocity are highly correlated with severity of diffuse brain injury (8,9), a fall from the lower ⅓ of the swing cycle may be more injurious than a fall from the apex of the arc. It is significant that five (cases 1, 3, 6, 17, and 18) of the six children who fell from swings were immediately unconscious, and the remaining child (case 16) was "groggy." This is consistent with diffuse brain injury arising from inertial forces. Only one child (case 10) who was not playing on a swing appears to have lost consciousness initially. This fall was not observed, and no conclusions about mechanism of injury can be made.

Twelve (67%) of the 18 cases in this series had a lucid interval (cases 2, 4, 5, 7–9, 11–16). However, in eight of these cases, the lucid interval was less than 1 hour (cases 2, 4, 5, 7, 8, 11, 12, and 16). One child (case 14) had a lucid interval of 2 days. This is by far the longest lucid interval ever described in a child. There is no precedent either in this series or in the papers cited (10,11) for a child who appears well and suddenly deteriorates many days or even weeks after apparently minor head trauma, a claim that is sometimes made in court.

The frequency of lucid intervals in this series correlates well with the infrequency of evidence of traumatic axonal injury on computed tomography scan or autopsy; it was recognized only in case 6 (5.6%), who fell from a swing. This is again in contrast with findings in children who have died of abusive head trauma (12,13) where traumatic axonal injury is common, particularly in the lower brainstem and upper spinal cord. With such findings, one would expect immediate loss of consciousness to be the rule rather than the exception.

The discussion of the literature concerning retinal hemorrhage and head injury in childhood is inadequate and misleading. The literature concerning the rarity of retinal hemorrhages in severe accidental head injury in childhood is extensive (14–16). These studies ably demonstrate that fewer than 2% of children with significant accidental head injuries have retinal hemorrhages when examined under optimal conditions by a pediatric ophthalmologist (3 of 209 in the combined series). In addition, the description of at least one of the retinal hemorrhages reported by Plunkett is probably erroneous. The report indicates that, in case 16, the hemorrhages extended to the periphery. Since the author stated elsewhere that none of the children had formal eye exams, it must be assumed that the fundus was examined by direct, rather than indirect, ophthalmoscopy. The periphery can be examined only by indirect ophthalmoscopy. If the physician responsible for describing the retinal hemorrhages was unaware of this, the validity of the rest of the description, in this case at least, is open to serious question. The author correctly identifies traumatic retinoschisis as a finding highly correlated with abusive head trauma. This finding was not described in any of the patients in the series. The paper cites the recommendation that children with serious head injury be examined by indirect ophthalmoscopy (17). Such examination would have clarified the extent and nature of retinal injury in this unusual group of patients.

The cited paper by Greenes and Schutzman (18) is misrepresented. This study of young children who were admitted to the hospital after detection of intracranial injuries revealed 19 children who had no neurologic symptoms, 7 of whom had subdural hematomas. However, Plunkett failed to indicate that 18 (95%) of the 19 children had a large scalp hematoma and skull fracture, injuries that are contact in origin. The visible scalp hematoma would have provided evidence to their caretakers that an injury had occurred. None of the 19 children deteriorated in hospital, although 1 had a post-traumatic seizure, which was well controlled with anticonvulsant therapy. All were discharged in good condition. Two-week follow-up of all cases revealed no subsequent deterioration.

This contrasts sharply with the dismal outcome noted in follow-up of infants who have suffered abusive head trauma. In Bonnier et al.'s study (19), 13 children diagnosed with abusive head trauma with a presumptive mechanism of shaking were followed for 5 to 13 years. Of these children, one died in the acute period, five were quadriplegic, blind and severely retarded, and one had delayed onset of epilepsy; all of these were diagnosed within the first year after injury. These children continued to be profoundly impaired during the follow-up period, and one suffered delayed death. At autopsy of this child, 10 separate tears were noted in the brainstem and cerebral hemispheres. Five of the six children

who were felt to be "normal" in the first year after injury had been identified as having significant disability by school age: two were hemiparetic; five were mentally retarded; and three had severe behavioral disorders. Only one still appeared to be normal. The extremely high morbidity and mortality are not unexpected with severe diffuse brain injuries caused by high inertial loads.

Plunkett's series adds to the knowledge concerning a rare but real event, childhood death from a short fall. However, it would be wrong and dangerous to draw conclusions from these deaths, which primarily resulted from serious contact forces, and apply those conclusions to a very different group of infants who die from shaking and/or shaking associated with impact, who have injuries primarily resulting from inertial forces. The differences between these two populations are well documented in the literature and are apparent in the findings in this report.

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REFERENCES

1. Plunkett J. Fatal pediatric head injuries caused by short-distance falls. *Am J Forensic Med Pathol* 2001;22:1–12.
2. Claydon SM. Fatal extradural hemorrhage following a fall from a baby bouncer. *Pediatr Emerg Care* 1996;12:432–4.
3. Reiber GD. Fatal falls in childhood. *Am J Forensic Med Pathol* 1993;14:201–7.
4. Home Playground Safety Tips: CPSC Document #323. Available at www.cpsc.gov.
5. Weber W. Experimental studies of skull fractures in infants. *Z Rechtsmed* 1984;92:87–94.
6. Weber W. Biomechanical fragility of the infant skull. *Z Rechtsmed* 1985;94:93–101.
7. Margulies SS, Thibault KL. Infant skull and suture properties: measurements and implications for mechanisms of pediatric brain injury. *J Biomech Eng* 2000;122:364–71.
8. Gennarelli TA, Thibault LE, Adams H, et al. Diffuse axonal injury and traumatic coma in the primate. *Ann Neurol* 1982; 12:564–74.
9. Gennarelli TA, Thibault LE. Biomechanics of acute subdural hematoma. *J Trauma* 1982;22:680–5.
10. Bruce DA, Alava A, Bilaniuk L, et al. Diffuse cerebral swelling following head injuries in children: the syndrome of "malignant brain edema." *J Neurosurg* 1981;54:170–8.
11. Snoek JW, Minderhoud JM, Wilmink JT. Delayed deterioration following mild head injury in children. *Brain* 1984; 107:15–36.
12. Shannon P, Smith CR, Deck J, et al. Axonal injury and the neuropathology of shaken baby syndrome. *Acta Neuropathol (Berl)* 1998;95:625–31.
13. Gleckman AM, Bell MD, Evans RJ, et al. Diffuse axonal injury in infants with nonaccidental craniocerebral trauma: enhanced detection by beta-amyloid precursor protein immunohistochemical staining. *Arch Pathol Lab Med* 1999; 123:146–51.
14. Elder JE, Taylor RG, Klug GL. Retinal haemorrhage in accidental head trauma in childhood. *J Paediatr Child Health* 1991;27:286–9.
15. Johnson DL, Braun D, Friendly D. Accidental head trauma and retinal hemorrhage. *Neurosurgery* 1993;33:231–5.
16. Duhaime AC, Alario AJ, Lewander WJ, et al. Head injury in very young children: mechanisms, injury types and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 1992;90:179–85.
17. Becker H, Gupta BK. Recognizing abusive head trauma in children. *JAMA* 1999;282:1421.
18. Greenes DS, Schutzman SA. Occult intracranial injury in infants. *Ann Emerg Med* 1998;32:680–6.
19. Bonnier C, Nassogne MC, Evrard P. Outcome and prognosis of whiplash shaken infant syndrome: late consequences after a symptom free interval. *Dev Med Child Neurol* 1995; 37:943–56.

To the Editor:

This communication is intended to address the biomechanical issues contained in the letter to the Editor by Betty Spivack, M.D., that critiqued the article by John Plunkett entitled, "Fatal Pediatric Head Injuries Caused by Short Distance Falls," which appeared in the recent issue of the American Journal of Forensic Medicine and Pathology. Its concepts are closely related to the work that she presented at the Third Shaken Baby Syndrome Symposium, with particular reference to the section labeled, "The biomechanics of Childhood Falls." The latter dissemination is either incorrect, incomplete, or out of context. Spivack's attempt to simplify this phenomenon fails because it both omits critical physical factors and misrepresents the motion. In particular, one issue emerging from both documents concerns the velocity of impact in falls from a swing, and another, on which this concept is predicated, utilizes the conservation of energy during the motion of the swing. Both representations are manifestly incorrect.

The motion of a rider falling off a moving swing is complex because of the possible independent movements of the jointed components of the body that may be produced voluntarily or involuntarily during both the phases while on the swing and during a subsequent free fall. In the final analysis, what matters with respect to the generation of trauma is the condition of impact with the ground and not what transpires during the fall. This depends on the velocity, direction, and constitution of the initial point of contact on the rider's body, the angular motion of the element of which this point is a part at that instant, the configuration of the rider at the time, the weights of the various components of the child, and the mechanical characteristics of the striking surfaces.

To discuss this issue in a proper context of mechanics, it is first necessary to define possible types of motions as well as the physical characteristics of swings and their riders. For simplicity, it will be assumed that the motion can be represented by a cross-section of the rider restricted to a single plane, a two-dimensional model called plane mo-

tion. Translation in this plane consists of a movement in which each point of the object under consideration travels the same path and has the same velocity and acceleration at a given instant of time. Rotation here occurs about an axis or a set of axes that are always perpendicular to the reference plane. In pure rotation, the velocity of any point of an object is equal to the instantaneous rotational (or angular) velocity multiplied by the perpendicular distance to the axis of rotation. In general, plane motion consists of a combination of translation and rotation. It is most frequently described by the translational motion of the center of gravity of the system and the rotation of its components about this position. This will entail an analysis of the motion of the independent parts of the body subject to the constraint that motion at the pin is the same for both joined segments. Here, the motion is most conveniently depicted with reference to the rider's center of gravity, whose path must be traced.

All swings hang by chains or similar suspensions attached to a set of bearings on the support structure at equal height from the ground. Most swings are arranged so that the seat is always perpendicular to the suspension, but in some cases there are bearings attaching the chain to the seat that permit either partial or full rotation with respect to the chain; in the last instance, the seat is maintained in a horizontal position. The fundamental motion of the seat and rider in these circumstances are totally different. In the first case, the system executes rotation about the axis through the two fixed suspension points, absent any twisting of the chains (which may very well occur, at least at certain times, and will produce three-dimensional motion). When the seat is permitted to rotate to some extent or even completely, the rider's movement is different. If the seat remains horizontal at all times, the motion of seat and rider is curvilinear translation. These and subsequent concepts may be found in any elementary text on dynamics, such as in (1) or (2).

All analyses of movement, other than the theory of relativity, which is irrelevant here, are based on Newton's second law of motion for an object treated as a geometric point (called a particle), but endowed with the property of mass, the quantity that resists motion in translation. Mass, in turn, is the weight of an object divided by the acceleration of gravity. Newton's second law states that force equals the product of mass and acceleration for a particle. All descriptions of causes of motion for bodies that do not qualify as a particle are derived from this law. Extension to rigid and deformable bodies, and to systems of connected bodies, can become very complicated. A particle, by definition, cannot execute rotation unless attached to another object, such as a string.

Consider first the conservation of energy, which, in part, underlies Spivack's hypothesis that the velocity of a child striking the ground in a fall from a swing depends only on the maximum height that the system has attained. Conservation of energy requires the sum of the kinetic energy (the energy of motion) and the potential energy (the height above a given reference plane) to be a constant. This implies the absence of external forces that do work on the system; but in a swing this is not the case. The rider "pumps" with his legs and body to make the seat rise to greater heights. This feeds energy into the system, drawn from the internal energy of the rider that the person can apply. Secondly, this "conservation" ignores the presence of both bearing and air friction; the former is substantial and, as is well known, soon brings a swing to rest if energy is not supplied either by the rider or a person on the ground pushing the swing. Any energy supplied by the rider can, in practice, never be instantaneously equal to the energy lost by friction.

For a system of completely rigid objects, whose configuration remains invariant, executing pure rotation about a fixed axis and ignoring the loss of energy because of friction permits the calculation of the velocity of any point of the system by equating the loss of energy of position to the gain of energy in motion. However, as already indicated, the rider is definitely not a rigid object (and perhaps the configuration of chain and seat may also not be regarded as totally fixed). In that case, the analysis of the motion of the system or its components becomes much more complex. It is first necessary to define the simplest possible model that will correctly define the movement of the system and its parts, particularly if the rider should fall from the swing.

It is not permissible to describe the motion of the rider as that of a mass point located at his or her center of gravity (a particle to which Newton's fundamental law applies without extension) because the dimensions of the rider are comparable to the distance traversed by the swing (for example, the earth in its circuit around the sun may be regarded as a point mass because its diameter, about 3,960 miles, is minuscule in comparison with the millions of miles traveled in one pass). The portion of Spivack's treatment dealing with the dependence of the velocity of impact solely upon the height of fall hints at such a point model, presumably the center of mass; she does not specify to which location on the rider her velocity refers, although it should designate the impact position. The rider cannot be considered a single rigid body, such as a match stick, because of

the ability to move the head and neck, upper and lower arms and legs, hands and feet, and even the torso, relative to the seat.

Thus, the simplest conceivable model appropriate for an analysis of the motion of a swing rider and a subsequent fall is a two-dimensional representation of plane motion, as described above, for a segmented object pinned at the joints, which minimally should include the head and neck junction, the shoulder and elbow joints, the hips and knees, and possibly the ankles and wrists. The analysis of such segmented motion has been established for some time and is detailed in (3), (4), and (5), but is too complicated and tedious to be described here, although it is not particularly difficult. It requires a knowledge of the weights and dimensions of the body segments. However, the actual motion is indeterminate here because of the need to precisely describe the configuration of the rider at the time of separation from the seat, the possible effects of a push of the swing during this event, and, most importantly, the unknown voluntary and/or involuntary movement of the child during the fall. Finally, the position of the center of gravity to the rider, which may be constantly changing at all times prior to impact, must be traced, preferably relative to some fixed point in space, although it can be referred to some anatomical feature of the rider.

At the instant of separation, the path of the center of mass of the rider will be a straight line perpendicular to the chains, with a constant linear velocity v_1 at an angle θ in the absence of gravity or a push from the swing, neglecting air resistance. All other points of the object will similarly retain the speed that they had just before departure at this same angle, resulting in a further rotation of the rider. However, gravitational attraction of all points of the person will contribute an additional vertical velocity component proportional to their distance of free fall, which must be added vectorially, never algebraically, to the velocity of any element of the rider because of the initial swing motion and subsequent deliberate or reflex action. The latter relative motion during the fall cannot be predicted; it can only be determined by photographic or other observation of the event. Thus, at impact, the total velocity of the center of mass, if its location remains constant within the configuration, depends on v_1, θ, and the velocity v_2, the latter being proportional to the square root of the height of fall at this point. Corresponding arguments apply to the total velocity of the contact point with the ground, whose location may be approximated from damage considerations. Its velocity component, because of swing and body actions, will practically never be in the vertical direction, so that its total value must also be determined vectorially and predicated on the configuration at impact. Hence, Spivack's contention that impact velocity does not depend on where a rider leaves the swing, only on total height attained, is invalid. Of course, the analysis presented here is a slightly simplified version of what may actually occur, because three-dimensional motions may be involved and an unknown amount of energy is pumped into the system by the controlled or involuntary independent motions of the rider. However, although extremely elementary, this representation at least depicts the action in a manner that is mechanically correct.

It should be mentioned that, if peak force experienced during the impact is a correlater of injury, the shorter the duration of contact (until the force is reduced to zero), the higher this peak force will be for a given set of conditions. This fact documents the obvious need for the most resilient possible surface under a swing, to decelerate a falling body more gradually and, hence, reduce the potential for injury. The analysis of the contact phenomenon is another story.

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REFERENCES

1. Meriam JL, and Kraige, LG. *Engineering Mechanics: Dynamics*, 3rd ed. New York: Wiley & Sons, 1992.
2. Berger SA, Goldsmith W, and Lewis ER. Biomechanics of solids. In: *Introduction to Bioengineering*. 2nd ed.: Oxford: Oxford University Press, Oxford, UK, 2000:1–100.
3. McHenry RR, and Naab KN. Computer simulation of the crash victim: a validation study. In: Proc. 10th Stapp Car Crash Conf., SAE Paper 660792, 1966.
4. Maltha J, and Wismans J, MADMO-Crash victim simulation–a computerized study. In: Proc. Vth Int. IRCOBI Conf. On the biomechanics of Impacts, 1–13; 1980.
5. Kane TR, and Levinson DA, Multibody dynamics, *J Appl Mech*, Trans. ASME, v. 50, 1071–8, 1983.

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Letters to the Editor

Retinal Hemorrhages: Evidence of Abuse or Abuse of Evidence?

To the Editor:

Sir, I would like to congratulate Dr. Plunkett and the *American Journal of Forensic Medicine and Pathology* for publishing his recent valuable contribution regarding infant head injury/shaken baby syndrome (1). No reasonable professional of any discipline wants to see malicious child homicide go undetected and unpunished. However, it is equally abhorrent that over-zealous investigation, refusal to study alternative explanations, or narrow-minded pursuit of blame for an infant's death can destroy the lives of the accused and their family, creating supplementary victims (2). This is particularly salient when the accused and the families of the accused are recently bereaved relatives of the deceased child. We have an inescapable duty to get this right! (3)

To date, published literature strongly supports the hypothesis that *only severe* traumatic forces cause the constellation of subdural hemorrhage, parenchymal brain injury, and retinal hemorrhage (4,5). However, in scientific terms, this is merely a *hypothesis* based on intrinsically imperfect epidemiology in the form of a case series, with case ascertainment and selection bias, and the formation of personal opinion. Experimental tests of the hypothesis via biomechanical or animal models have failed to confirm the necessity for severe force (6,7). However, these models are rightly criticized for not accurately reproducing the human state (2). Similarly, extrapolations from severe accidents, such as automobile accidents, while seemingly supporting the hypothesis (2,8), are themselves also imperfect biomechanical models of shaken baby/shaken impact syndrome. Biologic variation in responses is rarely considered (2) and professionals with voluminous experience working in this field will repeatedly encounter cases that just don't comfortably fit the mold. There may be little other suspicion of abuse, few other features of physical trauma, and minimal unequivocal evidence of traumatic brain injury despite the incongruous necessity for severe trauma. In addition, some adults consistently offer explanations involving minimal trauma or show bewilderment about the cause of the child's condition. Lawyers also candidly say that, while convinced of the guilt of some of their clients, they are convinced of the innocence of others. Nevertheless, the weight of best available medical and scientific knowledge is against them. Despite these difficulties, we are resolutely stuck with only *one* widely accepted hypothesis - a scenario very unusual in biologic systems in which multiple routes to a common end are normal.

Medical witnesses often refuse to challenge the weight of professional opinion, no matter how light, and prosecutors are only too willing to seize upon this convenience. Convictions add to the dogma that severe force is necessary and strengthens the self-fulfilling prophecy by adding "gray cases" to the spectrum of "proven black cases". Repeated "learned" publication of this dogmatic-received wisdom supports the potential myth, inexperienced professionals consume the hypothesis as *established fact*, and it becomes sacrilegious to challenge the hypothesis via contradictory observations or by offering alternative hypotheses (2). In this field more than others, we are more readily prone to alter the facts to fit the hypothesis rather than alter the hypothesis to fit the facts. For example, if the accused admits to severely harming the child, we tend to believe because this fits our hypothesis, but if the accused offers an alternative apparently innocuous explanation we discount the explanation because it doesn't fit the hypothesis (2). Published cases purporting to demonstrate less traumatic causes (9–12) are attacked for their anecdotal data and for overlooking the real, more sinister explanation (5). This "illogical inconsistency" overlooks scientific process and forgets that the requirement for severe shaking forces is no more than a *favored* but *unproven* hypothesis (13).

The main cause of bias and hindrance to progress is the fact that very few of these cases are verified by eyewitnesses (4). Dr. Plunkett's case series is very important because it involves verification of events by eyewitnesses and because the data was collected via an independent non-prosecutorial agency concerned with child safety and not conviction at all costs of a parent, guardian, or other child caregiver (1). The careful observations collated by Dr. Plunkett confirm that there are cases of innocent minor trauma that may mimic those of shaken baby syndrome. Dr. Plunkett employs sound biomechanical insight to challenge the accepted hypothesis

and to propose alternatives. His hypotheses may prove to be correct or wrong and are potentially as impotent as any others that have challenged the prevailing dogma. However, Plunkett's series is no weaker than those series that support the traditional hypothesis. Like other emerging publications questioning the causes and mechanisms of infantile brain injury (14,15), its most important function is to reopen debate, help us consider the status of our beliefs, and force us to seek the truth. In my own field of involvement, the pathologic interpretation of retinal hemorrhages, I am forced to note that Plunkett reports retinal hemorrhages in his series. This directly challenges the widely held view that retinal hemorrhages are "evidence of abuse" (5,16). To prevent retinal hemorrhages from being unwittingly abused as evidence, Dr. Plunkett's important paper must stimulate fresh thought and investigation into the spectrum of causes and mechanisms of infantile retinal hemorrhages (17).

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REFERENCES

1. Plunkett J. Fatal pediatric head injuries caused by short falls. *Am J Forensic Med Pathol* 2001;22:1–12.
2. Wilkins B. Head injury - abuse or accident? *Arch Dis Child* 1997;76(5):393–6;discussion 396–7.
3. Plunkett J. Shaken baby syndrome and the death of Matthew Eappen: a forensic pathologist's response. *Am J Forensic Med Pathol* 1999;20:17–21.
4. Duhaime AC, Christian CW, Rorke LB, Zimmerman RA. Nonaccidental head injury in infants–the "shaken-baby syndrome". *N Engl J Med* 1998;338:1822–9.
5. Child Abuse Working Party. Child abuse and the eye. The Ophthalmology. *Eye* 1999;13:3–10.
6. Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R. The shaken baby syndrome. A clinical, pathological, and biomechanical study. *J Neurosurg* 1987;66:409–15.
7. Ommaya AK, Faas F, Yarnell P. Whiplash injury and brain damage: an experimental study. *J Am Med Assoc* 1968;204:285–9.
8. Duhaime AC, Alario AJ, Lewander WJ, Schut L, Sutton LN, Seidl TS, et al Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 1992;90:179–85.
9. Goetting MG, Sowa B. Retinal hemorrhage after cardiopulmonary resuscitation in children: an etiologic reevaluation. *Pediatrics* 1990;85:585–8.
10. Aoki N, Masuzawa H. Infantile acute subdural hematoma. Clinical analysis of 26 cases. *J Neurosurg* 1984;61:273–80.
11. Aoki N, Masuzawa H. Subdural hematomas in abused children: report of six cases from Japan. *Neurosurgery* 1986;18:475–77.
12. Howard MA, Bell BA, Uttley D. The pathophysiology of infant subdural haematomas. *Br J Neurosurg* 1993;7:355–65.
13. David TJ. Shaken baby (shaken impact) syndrome: non-accidental head injury in infancy. *J R Soc Med* 1999;92:556–61.
14. Geddes JF, Vowles GH, Hackshaw AK, Nickols CD, Scott IS, WHitwell HL. Neuropathology of inflicted head injury in childre.2: Microscopic brain injury in infants. *Brain* 2001;124:in press.
15. Geddes JF, Hackshaw AK, Vowles GH, Nickols CD, WHitwell HL. Neuropathology of inflicted head injury in children. 1: Patterns of brain damage. *Brain* 2001;124:in press.
16. Green MA, Lieberman G, Milroy CM, Parsons MA. Ocular and cerebral trauma in non-accidental injury in infancy: underlying mechanisms and implications for paediatric practice [see comments]. *Br J Ophthalmol* 1996;80:282–7.
17. Tongue AC. The ophthalmologist's role in diagnosing child abuse. *Ophthalmology* 1991;98:1009–10.

Sudden Infant Death Syndrome

To the Editor:

I read with great interest the Letter to the Editor entitled, *SIDS Doesn't Exist*, by Drs. Sawaguchi and Nishida, which appeared in the June 2001 issue of the American Journal of Forensic Medicine and Pathology (1). I am a forensic pathologist who has, over the years, often stressed to students the true meaning of the term "SIDS". Sudden infant death syndrome is by definition a sudden natural death of an apparently healthy infant in whom no detectable cause of death can be found. This diagnosis is made only after all reasonable efforts to find the cause of death have been exhausted. It is, thus, not a wastebasket diagnosis and it does not mean that there is no cause of death, only that none can be determined by the forensic protocols and procedures used. It remains true that, in general, we recognize only that which is visible. It should therefore be clear that SIDS is not a cause of death. While it means that a cause of death is undetectable, it also implies that the death is a natural one.

I would like to note that the late Dr. Milton Helpern, my mentor, did in fact object to the use of the term "SIDS" because it could mistakenly convey the meaning of a medical condition, which it is not. For many years, he and his staff used the term "sudden unexplained death of infant," as can clearly be seen from the old records of the New York City Office of the Chief Medical Examiner. I believe that this is, in fact, a more accurate description of this phenomenon. The inherent danger of using the term "SIDS" can be seen in the case of a mentally deranged woman in Philadelphia who, at age 70, confessed to smothering eight of her young children decades ago. With no clear evidence to show otherwise, doctors and investigators had reluctantly attributed the deaths of these eight children—none of whom lived longer than 14 months—to sudden infant death syndrome (2). Thus, the danger of the ill-considered use of the term "SIDS" is clear.

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REFERENCES

1. Toshiko Sawaguchi et Hiroshi Nishida. SIDS Doesn't Exist. *Am J Forensic Med Pathol* 20001;21:211–2.
2. Mother admits she killed 8 of her children. The Associated Press, 1997.

Fatal Pediatric Head Injuries Caused by Short Distance Falls

To the Editor:

Dear Editor,

I read with interest the recent article "Fatal Pediatric Head Injuries Caused by Short-Distance Falls" (1)

The paper reports a retrospective chart review of 18 children who died from alleged accidental head injury. For six patients, data was available regarding an eye examination. Four of these patients had retinal hemorrhages (RH). The author uses these cases to suggest that perhaps RH must be interpreted with caution when evaluating a child for possible Shaken Baby Syndrome (SBS).

Unfortunately, "None of the children in this study had a formal retinal evaluation" and not enough details were given about the nature of the RH for us to make any determination about whether the findings are consistent with currently available published literature. One child had "extensive bilateral retinal and preretinal hemorrhage". Another had "bilateral retinal hemorrhage". A third had "extensive bilateral retinal, vitreous hemorrhage" in one eye and papilledema. The fourth child with RH was 10 years old and therefore not very applicable to the SBS age range. This child had "extensive bilateral confluent and stellate posterior, peripheral preretinal and subhyaloid hemorrhage". No details are given regarding post mortem microscopic examinations in any case.

If no child had a "formal" retinal examination (which I assume means ophthalmology consultation) then how are all the distinctions made about the types of RH? In our study of non-ophthalmologist retinal examinations (unpublished data) in a large cohort of SBS victims, not once did a non-ophthalmologist even attempt to make such distinctions. What does "extensive" mean? In my experience, what a non-ophthalmologist calls extensive might very well be well within the accepted posterior pole hemorrhages, which may be moderate in number (to me), and quite acceptable following severe accidental life-threatening head injury. What does "stellate" mean? I have never seen that word used to describe RH by an ophthalmologist or non-ophthalmologist. I cannot believe a non-ophthalmologist could distinguish between subhyaloid and preretinal hemorrhage. This is sometimes even hard for the ophthalmologist to do. And clearly, peripheral RH can only be seen with formal retinal examination using the indirect ophthalmoscope.

Interestingly, the incidence figures make perfect sense. To get these four cases, the author needed to search literally tens of thousands of records. In fact, one might even say that this article is proving that RH is even more rare than the literature already suggests. Posterior pole RH is seen in up to 3% of children who sustain severe accidental life-threatening head injury. In these cases, the history alone is almost always sufficient to differentiate the situation from SBS. Certainly, these are not falls out of the arms of adults or off of beds and sofas. In the current article, two of the cases with RH occurred on falls from swings. Even the author acknowledges that the height of swing falls "could not be accurately determined". In addition, with a child in motion, the velocity of the impact might be expected to be even higher than that from the height alone. Clearly, this is different than a "short-distance fall". The child with papilledema may have had RH secondary to the papilledema alone: a completely non-specific finding. Papilledema is uncommon in SBS.

So maybe this article reports an *extremely rare* circumstance where multiple factors coincide to allow RH (maybe even "extensive" RH) to occur following accidental trauma that is less than that which is usually needed (e.g. a motor vehicle accident) but still beyond common minor household falls. On the other hand, the paper may be completely inaccurate and misinterpreted if the eye examinations were not confirmed by an ophthalmologist.

Other than birth, SBS is by far and away the most common cause of RH in the first three years of life. Careful description of the number, types, and distribution of RH is essential in determining the specificity of the fundus picture. Much has gone into understanding RH in SBS and other conditions. Contrary to the author's suggestion that there is "scant objective evidence" to address this issue, the literature is replete with useful data. One review (2) has over 200 references. Rigorous application of the scientific method rather than incomplete anecdotal retrospective reports, is our best tool towards finding the truth.

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REFERENCES

1. Plunkett J. Fatal pediatric head injuries caused by short-distance falls. *Am J Forensic Med Pathol* 2001;22:1–12.
2. Levin AV. Retinal haemorrhage and child abuse. In: David TJ, (ed). *Recent Advances in Paediatrics*, no. 18. London: Churchill Livingstone, 2000:151–219.

Author's Response to Drs. Spivack and Levin

To the Editor:

I thank Drs. Spivack (1) and Levin for their letters and have only a few comments.

The initial manuscript was almost 50% longer than the final version. The published version, at more than 6000 words, was still very long by any standard. It is difficult to include every detail for every case in a series this large and still make the article readable. Ideally, this study would have been prospective and would have involved every death investigation jurisdiction in the United States, with a detailed protocol for evaluating and documenting each death. However, this could not be done, in part because there has been no impetus to do so since these types of injuries and deaths "cannot and do not occur".

The series cannot be used to establish either the commonality or the rarity of death from a given event for the reasons stated in the "Limitations" and the "Conclusions"(2).

Weber's papers (3-4) are actual scientific studies based on a given mechanical input (a gravitational fall of 82 cm to a variety of surfaces) and a measured or observed output (skull fracture) under controlled conditions. My observations should not be used to either confirm or deny the validity or applicability of his observation.

The mechanism (with few exceptions) for an ultimately fatal brain injury in an infant or child is not multifocal traumatic axonal injury (DAI), but focal traumatic axonal injury with secondary anoxia and/or cerebral edema (5-8) or an acute subdural hematoma. The focal injury may be a contact-induced cortical contusion, cortical laceration from a skull fracture, or superficial brainstem contusion caused either by hyperextension or by movement of the brainstem through the foramen magnum (that may occur secondary to mass movement of the brain caused by deformation of the skull during impact) (9). One would not predict nor does one usually find DAI in circumstances where the mechanism is a short-distance fall or a "slam". (The physics of a short-distance fall and a "slam" are identical).

There is not a single reference to "shaken baby syndrome" or "SBS" in the text of the article. Dr. Levin states that I "suggest perhaps that RH must be interpreted with caution when evaluating a child for possible shaken baby syndrome (SBS)". My actual "conclusion" regarding retinal hemorrhage is, "The characteristics of the bleeding cannot be used to determine the ultimate cause."

The eye examinations were performed by a Board-certified neurosurgeon with 25 years of experience (case #16) and by Board-certified pediatric intensivists with 5–15 years of experience (cases #4, 5 and 6). I discussed the medical record entries with each of these physicians. The descriptions in the article are theirs, not mine. If someone believes that these physicians were not qualified to make the observations and conclusions that they did, then so be it.

Although this study should not be used to determine the incidence of retinal hemorrhage associated with head impact from a fall (for the reasons stated in the "limitations"), it is not true that "tens of thousands" of cases were searched to identify these four. The correct number is *18*, and of these, only *six* had fundoscopic examination performed.

There is no evidence that "shaking" can cause retinal hemorrhage or that the mechanics of an impact from a "slam" are somehow different from those of a "fall". The conclusion that they are different is tautology, not science. If someone has a theory for a cause of retinal hemorrhage different from those that I discuss in the study, then please do the appropriate experiments and prove it. Reiterating the hypothesis and stating that it is "widely and generally accepted, published," and authenticated by "vast clinical experience" does not make it true.

I hope that my study encourages us to re-examine our concepts regarding traumatic brain injury (TBI) and the relative importance of inertial or impulsive loading (whiplash) and contact. Dr. Caffey's "theory," accepted for almost 30 years, taught in medical schools, approved as an ICDA-9 "codable disease" and testified to as "truth" in court, is based on a misinterpretation of early pioneering experiments performed for the automotive and space industry. Ommaya (10) published a landmark study in 1968 showing that TBI could be produced in rhesus monkeys by acceleration of the head alone (with the midneck as a fulcrum) and no contact. However, the level of acceleration he used to cause these injuries was 10,000–100,000 r/s^2, with the lower limit being the *concussion* threshold. (Ten thousand r/s^2 at a radius of 6 inches is 5,000 f/s^2 or 156 G's). Caffey called Ommaya after his (Caffey's) 1972 article (11) was published and discussed it with him (12). Ommaya told him that he (Caffey) was misinterpreting his (Ommaya's) studies, but Caffey either didn't understand or forgot to tell us. This misinterpretation is repeated in Caffey's 1974 article (13). And here we are today. A WWII paratrooper aphorism concerning chute-deployment failure says it best: "It is not the fall that kills you. It's when you hit the ground."

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REFERENCES

1. Spivack B. Fatal pediatric head injuries caused by short-distance falls. *Am J Forens Med Pathol*; 22:332–34.
2. Plunkett J. Fatal pediatric head injuries caused by short-distance falls. *Am J Forensic Med Pathol* 2001;22:1–12.
3. Weber W. Experimental studies of skull fractures in infants. *Z Rechtsmed* 1984;92:87–94.

4. Weber W. Biomechanical fragility of the infant skull. *Z Rechtsmed* 1985;94: 93–101.
5. Oehmichen M, Meibner C, Schmidt V, Pedal I, Konig HG, et al. Axonal injury—A diagnostic tool in forensic neuropathology? A review. *Forensic Sci Int* 1998;95:67–83.
6. Geddes JF, Whitwell HL, Graham DI. Traumatic axonal injury: practical issues for diagnosis in medicolegal cases. *Neuropathol Appl Neurobiol* 2000;26:105–16.
7. Geddes JF, Hackshaw AK, Vowles GH, Nickols CD, Whitwell HL. Neuropathology of inflicted head injury in children. I. Patterns of brain damage. *Brain* 2001;124:1290–98.
8. Geddes JF, Vowles GH, Hackshaw AK, Nickols CD, Scott IS, et al. Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants. *Brain* 2001:124:1299–1306.
9. Margulies SS, Thibault KL. nfant skull and suture properties: measurements and implications for mechanism of pediatric brain injury. *J Biomech Eng* 2000;122:364–71.
10. Ommaya AK, Faas F, Yarnell P. Whiplash injury and brain damage. *JAMA* 1968;204:285–89.
11. Caffey J. On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child* 1972;124:161–70.
12. Ommaya AK. Personal communication. April 27, 2001.
13. Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleeding, linked with residual permanent brain damage and mental retardation. *Pediatrics* 1974; 54:396-403.

Death Resulting from Asthma Associated with Sertraline "Overdose"

To the Editor:

In their report, Carson et al. (1) speculate that the asthma-related death of a young woman might have been caused by serotonin syndrome, in turn caused by a sertraline overdose.

In support of an overdose, they state that the blood level of sertraline, 620 ng/ml, was "very high." However, they do not mention the analytical method used or the sampling site of the blood, nor do they use relevant postmortem reference levels.

To determine whether a postmortem drug concentration is high, reference data are needed. The authors mention a steady-state concentration range of 30–200 ng/ml in persons receiving "therapeutic dosage regimens," but they do not give a reference. Although this is approximately the range that Gupta and Dziurdy (2) report in their clinical study, such data are usually not suitable for comparison with postmortem levels, particularly because of postmortem drug redistribution (3). In addition, even postmortem reference data may be unsuitable if the sampling site is unknown or if the procedures and methods are not specified.

In a compilation of postmortem analytical results, Druid and Holmgren (4) have tried to overcome some of these obstacles for several drugs. First, only femoral blood levels are used. Second, levels are provided both for lethal intoxications and for carefully selected cases in which the cause of death with certainty was not related to intoxication, and in which the victims were not incapacitated by drugs immediately before death. Third, as to the fatal intoxications, levels are given separately for cases related to intoxication with one substance only and for cases in which other substances or conditions may have contributed to death.

In a recent report, the same strategy was used to evaluate additional drugs, including some of the newer antidepressants (5). A total of 211 cases in which sertraline was quantified in femoral blood were reviewed. There were no sertraline-only deaths. In cases in which sertraline intoxication in combination with other factors was considered to have caused the death, the median concentration was 2.2 (10th and 90th percentiles 1.3 and 3.6) μg/g blood (n = 11), in comparison with 0.1 (0.1 and 0.4) μg/g blood (n = 61) in nonpoisoning deaths. The lack of sertraline-only deaths in this Swedish report may also suggest a low toxicity in comparison with other selective serotonin reuptake inhibitor drugs when the prescription data are taken into account (6).

Although the reported concentration of 0.62 μg/ml exceeds the median level in the "controls" of this report, it is still lower than the concentrations found in the cases in which sertraline was considered to have been a contributory cause of death. It is therefore hardly justified to say that the level is "very high." Moreover, the origin of the blood sample is not stated. If the blood sample was collected from the heart, it is even more likely that the concentration actually was not much elevated ante mortem, given that leakage and redistribution of the drug from other tissues with high concentrations could have occurred. In a recent study by Goeringer et al. (7), based at least partly on peripheral blood, the authors concluded that sertraline appeared to be a primary contributory factor to the death at levels above 1.5 mg/L, thus also significantly higher than the reported level of 0.62 mg/L.

The authors are apparently unaware that they do provide one clue in support of a *recent* intake: the parent drug–to-metabolite ratio, which is 1.9. This figure is higher than the median (0.6) reported by Druid and Holmgren (5) for controls, but it does not necessarily confirm a *large* intake. The ratio might also be influenced by various pharmacokinetic and pharmacogenetic factors.

In conclusion, the suggestion that the asthma attack was precipitated by a sertraline overdose leading to very high sertraline concentrations thus remains speculative.

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Letters to the Editor

Chronic Lead Poisoning: Induced Psychosis in an Adult?

To the Editor:

I recently had a case of a 35-year-old healthy white man with no history of physical or mental illness who, for 1 1/2 years, had been sanding the paint off a 125-year-old ranch house. He worked both indoors and outdoors, rarely using a mask. Two months before his death, he experienced chest pain and tingling in his fingertips. His physician ruled out cardiac problems and prescribed alprazolam, thinking his symptoms were secondary to stress. Three days later, he became extremely anxious, restless, delusional, and paranoid, and was unable to sleep.

One month before death, he attempted suicide with his automobile but survived. During his hospitalization, he was anemic (hemoglobin of 12.1 g/dL) and had 1+ proteinuria and 3+ hematuria. He became very depressed and was started on an antidepressant. A month later, he committed suicide by way of an intraoral gunshot wound. The only unexpected finding at autopsy was a blood lead level of 51.3 μg/dL. The last exposure to the ranch house was 8 weeks before death.

A brief review of the literature suggests that it is possible that the decedent's psychiatric disturbances were caused by chronic lead poisoning. The primary routes of lead absorption are ingestion and inhalation. Homes built before 1940 used lead-based paints on both the exterior and interior surfaces. A tiny flake from an old house can contain up to 100 mg of lead (1). Accumulation and toxicity occur if more than 0.5 mg/day is absorbed. Chronic lead poisoning is more common than acute lead poisoning and produces a variety of signs and symptoms (2).

Lead interferes with the synthesis of heme, and consequently a hypochromic normocytic anemia develops along with a compensatory reticulocytosis and basophilic stippling. Gastrointestinal symptoms are constipation, abdominal pain ("lead colic"), anorexia, weight loss, vomiting, and metallic taste. There is damage to the proximal tubules of the kidney, with subsequent proteinuria, hematuria, and oliguria. Thus, the anemia, proteinuria, and hematuria during this individual's hospitalization for attempted suicide are consistent with hematopoietic and renal injury caused by chronic lead exposure.

Relative to the nervous system, patients with chronic lead poisoning manifest encephalopathy (including irritability, fatigue, restlessness, and insomnia followed by delirium, convulsions, and coma) as well as peripheral neuropathy with tingling in the hands and feet, and wrist and foot drop. Baker et al. performed a prospective study of lead neurotoxicity in foundry workers and referents in 1983, finding that there was an increased rate of depression, confusion, anger, fatigue, and tension in workers with blood lead levels over 40 μg/dL (3).

Several factors support the possibility that the decedent in this case had chronic lead poisoning. The blood level at autopsy was 51.3 μg/dL. A blood lead level above 5–10 μg/dL indicates exposure, and levels above 40 μg/dL are considered toxic. One's risk of encephalopathy is great with levels above 80 μg/dL, and it is considered an emergency with levels above 100 μg/dL (2). Death was 8 weeks after his last exposure to the lead, and given the fact that the t½ of lead in blood is 1 to 3 months, his lead level at the onset of his psychiatric symptoms was probably above 100 μg/dL.

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REFERENCES

1. Gossel TA, Bricker JD. *Principles of Clinical Toxicology*, 2nd ed. New York: Raven Press, 1990:173–7.
2. Dreisbach RH, Robertson WO. *Handbook of Poisoning: Prevention, Diagnosis and Treatment*, 12th ed. East Norwalk, Connecticut: Appleton and Lange, 1987;230–6.
3. Baker EL, Feldman RG, White RF, Harley JP. The role of occupational lead exposure in the genesis of psychiatric and behavioral disturbances. *Acta Psychiatr Scand Suppl* 1983;303:38–48.

Fatal Pediatric Head Injuries Caused by Short Distance Falls

To the Editor:

In the recent article by Plunkett entitled "Fatal pediatric head injuries caused by short distance falls" (1) he states, based on a literature review, "many physicians believe that a simple fall cannot cause serious injury or

death, that a lucid interval does not exist in an ultimately fatal pediatric head injury, and that retinal hemorrhage is highly suggestive if not diagnostic for inflicted trauma." Our review of the literature referenced by Plunkett shows his opening statements to be an inaccurate and misleading summation of the conclusions drawn by the authors of the work cited.

Here are a few examples. Williams' (2) concluded: "infants and small children are relatively resistant to injuries from free falls, and falls of less than 10 feet are unlikely to produce serious or life-threatening injury." Willman et al. (3) concluded: "unless an epidural hematoma is present, children who die from blunt head injuries probably do not experience lucid intervals." and Rao et al. (4) concluded: "if intraoccular hemorrhage is seen, the possibility of child abuse should be considered."

All of the authors recognize, as we do, that there are no absolutes in regard to head injuries in children and refrain from using Plunkett's absolutist vocabulary of "can not, does not, and is diagnostic for," especially when it concerns the issue of accidental versus inflicted trauma in children. As forensic pathologists we must address each case without prejudice, paying particular attention to anamnestic-anatomic disharmony, cognizant that the issue is seldom black or white, and admitting that impossible situations are few and far between.

Some of the limitations of the study have been cited within the text of the article but must be emphasized, with special attention to children under the age of 5. The biophysics of head injury in children from newborn to 60 months changes fundamentally, and most controversies involve children from 0 to 36 months of age. Six of the 18 falls were not witnessed, including 5 of the 10 falls of children 5 years of age or younger. In this study, no child less than 23 months of age had a witnessed fall. Seven of the 18 falls were not followed by a full autopsy, including 5 of the 10 falls of children 5 years of age or younger. Although physical and radiologic examinations are helpful in assessing injury, they cannot fully answer questions as to anamnestic-anatomic disharmony, occult injuries, or undiagnosed disease processes. Cases without autopsy results must be viewed with circumspection.

The evaluation of the history of a lucid interval in cases of lethal head injury is especially important in children under the age of 5, whose injuries are often unwitnessed. In this study, there are 4 witnessed cases of children under the age of 5 years (Cases 5, 6, 8, and 9), who were evaluated with autopsy and forensic pathologist directed-death investigation system examinations that deserve further discussion. The lucid intervals of the witnessed cases ranged from none to 3 hours. In the case with no lucid interval (Case 6), there was a lateral blunt impact injury of the head, focal hemorrhage in the right posterior midbrain and pons consistent with diffuse axonal injury, a small subdural hemorrhage, and cerebral edema with herniation after at least a 4-hour survival period. In two of the cases there were lucid intervals of a few minutes (Cases 5 and 8). In the first case, there was a blunt impact injury of the right side of the forehead with large subdural hematoma requiring evacuation. Cerebral edema and herniation developed after at least a 12-hour survival period. In the second case, there was a complex fracture of the frontal-temporal bones associated with cortical contusions, epidural and subdural hemorrhages, and the development of malignant cerebral edema with herniation on the second hospital day. The child died on the third hospital day. In the case with a lucid interval of 3 hours (Case 9), there was an epidural hematoma without skull fracture or blunt impact injury of the head, and the child died 10 days after admission to the hospital. In each case, the lucid intervals were consistent with the neuropathologic findings at the time of autopsy.

The height of the fall could be determined in only 10 of 18 cases. However, Plunkett's definition of the height of the fall as "the distance of the closest body part from the ground at the beginning of the fall" would mean that a fall from standing height would be interpreted as a height of zero! Obviously, the determination of height of the fall in assessing head injury cases would better be defined as "the distance of the head from the ground at the beginning of the fall," which would increase the distance determination in these cases.

The vast majority of children (14 of 18) had evidence of blunt impact to the head by radiologic evaluation, autopsy documentation, or witnessed reports that were consistent with the given scenarios. One case had no reports (Case 11). Of the three children with no evidence of blunt impact to the head (Cases 3, 9, and 15), two had large subdural hematomas, and the third had an epidural hematoma. This study suggests that lethal impacts to the head rarely leave no evidence of an impact. None of the children without reported head impact injury had retinal hemorrhages.

We agree with Plunkett that deformation and internal angular acceleration of the skull and brain caused by the impact produce injury. Although no specific mathematical analysis was applied to these cases, it also may be helpful to consider that in each of these cases the head was in motion preceding impact because of the nature of the accelerating and/or centrifugal forces of the playground equipment that the child was using (swings, seesaw, horizontal ladders) or because of a fall from losing balance. In falls from losing balance, the center of gravity becomes displaced with respect to the feet, and the force of gravity rotates the body in the direction of the displacement. In these rotational falls, the head's acceleration exceeds the acceleration because of gravity (5). Medical examiners frequently are presented with histories that are similar to the type of falls studied by

Lyons and Oates (6) rather than the histories that are presented in Plunkett's study.

This review of more than 75,633 entries of childhood falls associated with playground equipment in the databases of the U.S. Consumer Protection Agency during an 11½-year period has served to document the *extremely rare* occurrence of lethal head and neck injury in children in *unique scenarios*. By design, these databases are biased toward the collection of data regarding severely injured children, and the total number of entries in the databases cannot approximate the millions of childhood falls of short distances *not* reported to the Consumer Protection Agency during that same 11½-year period in association with playground equipment or otherwise. The study is limited in its evaluation of infants and children under the age of 5 because there are no witnessed cases in children under the age of 23 months. Evaluation of the only four witnessed cases of children under 5 years of age with findings documented by autopsy and forensic pathologist directed-death investigation system examinations showed that lucid intervals are consistent with neuropathologic findings at the time of autopsy. The determination of height of fall in this study is difficult to interpret because it does not reflect the height of the head at the time of the fall. There were no cases without blunt impact injury of the head that showed small subdural hemorrhages and retinal hemorrhages. Certainly a history given by a caregiver that a child may have fallen must be evaluated, but there is no substitution for a thorough postmortem examination, including medicolegal investigation and the interview of witnesses.

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REFERENCES

1. Plunkett J. Fatal pediatric head injuries caused by short distance falls. *Am j Forensic Med Pathol* 2001;22:1–22.
2. Williams RA. Injuries in infants and small children resulting from witnessed and corroborated free falls. *J Trauma* 1991;31: 1350–2.
3. Willman KY, Bank DE, Senac M, Chadwick DL. Restricting the time of injury in fatal inflicted head injury. *Child Abuse Negl* 1997;21:929–40.
4. Rao N, Smith RE, Choi H, et al. Autopsy findings in the eyes of fourteen fatally abused children. *Forensic Sci Int* 1988;39: 293–9.
5. Dawson SL, Hirsch CS, Lucas FV, Sebek BA. The contrecoup phenomenon: Reappraisal of a classic problem. *Hum Pathol* 1980;2:155–66.
6. Lyons TJ, Oates RK. Falling out of bed: Relatively benign occurrence. *Pediatrics* 1993;92:125–7.

Author's Response to Fatal Pediatric Head Injuries Caused by Short Distance Falls

To the Editor:

I thank Dr. Shaber and colleagues for their letter. I agree with many of their points regarding the limitations of the study. However, I have a few additional comments.

I do not think that my opening statement is "inaccurate and misleading" or unfairly characterizes the conclusions in the references that I cite. For example, Schaber et al. state that Williams (1) concludes: "Infants and small children are relatively resistant to injuries from free falls, and falls of less than 10 feet are unlikely to produce serious or life-threatening injury." This "conclusion" is from the abstract. The text of the article states: "That severe injuries and deaths from falls of 5 feet or less only occurred in the uncorroborated group leads one to suspect that many if not all of the injuries attributed to falls of low height represent child abuse." Likewise, when Willman et al. (2) conclude that "Unless an epidural hematoma is present, children who die from blunt head injuries probably do not experience lucid intervals" they also state: "The results of this study suggest that a fatal HI [head injury] that does not involve an epidural hemorrhage must have occurred after the last known time that the child exhibited normal behavior," "For an older child . . . the time of the injury event in a fatal HI without an epidural hemorrhage can be restricted to after a confirmed period of such normal behavior," and "Excepting cases involving epidural hematomas, the time of injury in a fatal head injury case can be restricted to after the last confirmed period of normal consciousness for the child". [I comment on the Willman et al. study in a letter published in 1998 (3).]

Shaber's assertion that "The biophysics of head injury and children from newborn to 60 months changes fundamentally" lacks an evidentiary basis. The brain, scalp, and skull of a newborn are *not* the same as the head and neck unit of an adult. Scaling commonly used for the automotive industry may *not* apply to the neonate or infant (4). The developmental anatomy and physiology are certainly different, but where is the cutoff, and where is the *biophysical* evidence to support it? There is only one published study of the biomechanical failure characteristics of the infant skull (5). There are only three published studies of experimentally produced infant skull fractures (6–8). There is one published reference concerning the concussion threshold in the pediatric age group (9). There are no other studies evaluating the failure thresholds or the differential biophysics of infant, toddler, or pediatric head injury, whether for a 3-month-old, 3-year-old, or 13-year-old.

I agree that "cases without autopsy results must be viewed with circumspection." It would have been invaluable if complete postmortem examinations and formal neuropathology evaluation, including microscopic examination, had been performed in all of the deaths. However, please note that four of the seven deaths in which an autopsy was not performed occurred in a jurisdiction directed by a board-certified forensic pathologist. (This is not a criticism; I am merely emphasizing that one can rarely anticipate all of the questions that may be asked in the future about a death that seems "routine" or "adequately and fully documented" today.)

I agree that the documentation of the height of the fall "would be better defined" as suggested. The definition I used allows an initial comparison of the cases in my study with those from other cited articles, none of which has *any* definition for "height of the fall" and wherein it must be assumed, correctly or incorrectly, that the "height" is the height of the object from which the person fell (10–14). However, none of the other studies describes the position of the body at the beginning of the fall, the initial velocity, the part of the body that first struck the ground, or the characteristics of the impact surface. These cited studies do not allow even a kinematic analysis, let alone a kinetic evaluation. Further, using "head above the ground" does not elucidate the mechanism for injury and only allows a calculation of the approximate impact velocity. For example, knowing the distance of the head above the ground does not allow one to evaluate momentum (mv), energy ($\frac{1}{2}mv^2$), force (ma), deformation and strain of the scalp, skull, or brain, etc.

Each of the falls in this study involved an external rotational component. None was purely "translational," and I discussed the mathematical constructs for analysis of the falls in the Appendix. However, whereas external angular motion during a fall may change the impact velocity from that calculated for simple translational motion (greater or less, depending on whether the vectors are additive or subtractive), angular motion does not significantly contribute to "inertial loading." What happens during the impact, not during the fall, determines the outcome.

Dr. Shaber states that "This review of more than 75,633 entries of childhood falls associated with playground equipment . . . document(s) the *extremely rare* occurrence of lethal head and neck injury . . . in *unique scenarios*" and "By design, these databases are biased toward collection of data regarding severely injured children." Her contention is a mischaracterization of the database (15–18). Certainly, the death certificate (DC) file is biased. However, only 4 of the 47 cases in the CPSC DC file involved a fall (Cases 1, 5, 6, and 9). The others involved events described in the first paragraph of the Results. The incident report (IR) file includes many entries in which there was *no* injury, only the potential for injury. The NEISS file (more than 75,000 entries) contains reports of emergency room visits in the United States hospitals participating during the study period, and includes all incidences, not simply falls (as I discussed in the Introduction, Methods, and Results). More than 98% of the entries in the NEISS file involved an injury not requiring hospitalization. It is important to know that deaths such as I reported do occur, and it is important to understand why they happen. Whether the events are common or rare is not.

Finally, I agree that there is "no substitution for a thorough postmortem examination including medicolegal investigation and interview of witnesses." However, I would add that this investigation must be founded upon the ability to critically evaluate the evidentiary bases for opinions and conclusions, and upon an accurate, complete, and current understanding of the biomechanics of head injury.

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REFERENCES

1. Williams RA. Injuries in infants and small children resulting from witnessed and corroborated free falls. *J Trauma* 1991;31:1350–2.
2. Willman KY, Bank DE, Senac M, Chadwick DL. Restricting the time of injury in fatal inflicted head trauma. *Child Abuse Negl* 1997;21:929–40.
3. Plunkett J. Restricting the time of injury in fatal inflicted head trauma [Letter]. *Child Abuse Negl* 1998;22:943–4.
4. Irwin A, Mertz HJ. Biomechanical basis for the CRABI and Hybrid III child dummies. New York: The Society for Automotive Engineers, Inc., Document #973317, 1997:261–72.
5. Margulies SS, Thibault KL. Infant skull and suture properties: Measurements and implications for biomechanics of pediatric brain injuries. *J Biomech Engin* 2000;122:364–71.
6. Taylor AS. Marks of violence on the head: Fractures of the skull. In Taylor AS, Hartshorne E, eds. *Medical jurisprudence.* Philadelphia: Blanchard & Lea, 1856:366–70.
7. Weber W. Experimental studies of skull fractures in infants. *Z Rechtsmed* 1984;92:87–94.
8. Weber W. Biomechanical fragility of the infant skull. *Z Rechtsmed* 1985;94:93–101.
9. Sturtz G. Biomechanical data of children. New York: The Society of Automotive Engineers, Inc. Document #801313, 1980;513–59.
10. Helfer RE, Slovis TL, Black M. Injuries resulting when small children fall out of bed. *Pediatrics* 1977;60:533–5.
11. Hall JR, Reyes HM, Horvat M, Meller JL, Stein R. The mortality of childhood falls. *J Trauma* 1989;29:1273–5.
12. Chadwick DL, Chin S, Salerno C, Landsverk J, Kitchen L. Deaths from falls in children: How far is fatal? *J Trauma* 1991;31:1353–5.
13. Lyons TL, Oates RK. Falling out of bed: A relatively benign occurrence. *Pediatrics* 1993;92:125–7.
14. Reibur GD. Fatal falls in childhood: How far must children fall to sustain fatal head injury? Report of cases and review of the literature. *Am J Forensic Med Pathol* 1993;14:201–7.
15. National Injury Information Clearinghouse. US Consumer Products Safety Commission, 1997. Available at: www.cpsc.gov/about/clrnghse.html

16. Consumer Product Safety Review 4:#2:3–7, 1999. Available at: www.cpsc.gov/cpscpub/pubs/cpsr.html
17. A description of the injury or potential injury incident database (IPII). Division of Hazard and Injury Data Systems. US Consumer Products Safety Commission, 1997. Available at: www.cpsc.gov/about/guide.html#OIPA
18. A description of the in-depth investigation database (INDP), fiscal year 1987–fiscal year 1991. Division of Hazard and Injury Data Systems. US Consumer Products Safety Commission, 1992. Available at: www.cpsc.gov/about/guide.html#OIPA .

Symptoms Following Head Injury

To the Editor:

A recent proposed position paper regarding pediatric nonaccidental abuse head injury would allege that we may depend on symptoms to appear immediately upon injury (1). I have a concern about that claim, which I will explain with the following case. CASE REPORT

A 13-month-old Hispanic girl was brought to the University of Wisconsin Hospital on the morning of September 18, 1999. The complaint was of vomiting that had lasted for 24 hours. She was described as irritable, sleepy, and vomiting. In our emergency room she was noted to have extensive bruises on the cheeks, chest, back, and arms; the mother attributed these to bites by a 3-year-old housemate. She was admitted and given intravenous fluids. She was sedated with pentothal followed by head computed tomography, which was negative. She was then admitted to the pediatric ward. The resident who saw her described her in the chart and in discussion as being fussy and clingy, but interactive and responsive. Because of the numerous bruises, the police were notified and took pictures.

At about 2:00 the following morning, a nurse coming in to care for the child noted that she had decreased respirations. It was then shown that she was unresponsive and had a right dilated unreactive pupil with a sluggish left pupil. She was taken emergently to the pediatric intensive care unit, where she was intubated and given mannitol. A subsequent computed tomography scan showed very poor differentiation of gray/white matter interface. A Codman catheter was placed and then replaced with a ventriculostomy tube after an intracranial pressure of 21 mm Hg was noted.

On the evening of the day after admission, a cerebral blood flow study showed no cerebral blood flow. She was pronounced brain dead.

In the interim, her mother had fled town and has not been found since.

An autopsy was done on September 20, 1999. This showed hemorrhage in the left optic nerve sheath and left retinal hemorrhages as well as marked cerebral edema and thin widespread subdural hemorrhage. Diffuse axon injury was demonstrated with amyloid precursor protein antibody.

My point is that the child did have some symptoms, but clearly the severe intracranial injury symptoms, which were confirmed on repeat computed tomography and autopsy, were delayed for several hours, during which time she was under our view and review in the hospital. Others have noted similar problems (2).

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REFERENCES

1. Case ME, Graham MA, Handy TC, Jentzen JM, Monteleone JA. Position paper on fatal abusive head injury in infants and young children. *Am J Forensic Med Pathol* 2001;23:112–22.
2. Gilliland MGF. Interval duration between injury and severe symptoms in non-accidental head trauma in infants and young children. J Forensic Sci 1998;43:723–5.

Author's Response

To the Editor:

In response to Dr. Robert Huntington's letter in which he expresses concern about the Position Paper's position on interval from injury to symptoms in young children with abusive head injuries, I would make the following comments. Dr. Huntington describes a child who is noted to be "irritable, sleepy, and vomiting" on admission. The Position Paper states, "Symptoms demonstrated by these severely injured children include an immediate decrease in the level of consciousness (either lethargy or unconsciousness)." Lethargy is defined as a condition of drowsiness or indifference *(Dorland's Illustrated Medical Dictionary)*. The sleepiness in this child is the neurologic symptom that marks the time at which the injury to this child occurred. It indicates a decrease in the level of consciousness. After the child was in the hospital several hours, she showed signs of increased intracranial pressure (right dilated pupil and sluggish left pupil) and went on to brain death. Dr. Huntington remarks that the severe intracranial injury symptoms were delayed for several hours. The symptoms to which he is referring were not the initial symptoms of injury but those related to increased intracranial pressure. The brain injury that precipitated this course of events (diffuse axonal injury) occurred when the child first became neurologically symptomatic. Brain swelling followed the diffuse axonal injury and resulted in increasing intracranial pressure and eventually brain death. Certainly, the child's neurologic symptoms changed with time, reflecting this changing neuropathology. It is the initial neurologic change that marks the time of injury.

The article by Gilliland that Dr. Huntington mentions is not helpful in elucidating the interval from injury to symptoms. The cases reported in that article relied on histories from possibly biased witnesses (caregivers who

might have injured a child) and took at face value the time intervals provided in each case. Dr. Gilliland noted this problem in her article, stating, "It should be noted that in all of the cases where information was supplied by someone other than the perpetrator, the child was not normal during the interval."

The claim that a young child has been fine for hours after a fatal head injury was inflicted and then suddenly developed symptoms is a claim that has no support from legitimate or mainstream medicine. When a child has suffered a serious acceleration injury to the brain that will result in long-term neurologic impairment or cause death, the so-called lucid interval is a fiction. The change from "fine" to "not fine" may be lethargy or it may be unresponsiveness, but it is a neurologic change, and it occurs at the time of injury.

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Classical mistakes in forensic pathology*

These are several unique features of the mistakes that are peculiar to the performance of medicolegal autopsies. One is the frequency with which mistakes are made by good pathologists. Another is the frequency with which a seemingly trivial error turns out to have disastrous consequences. Perhaps fewer mistakes would be made if there were more widespread appreciation of what constitutes a mistake in the performance of a medicolegal autopsy, and why it is a mistake.

The factual material upon which this discussion is based is derived from several sources. First are the mistakes that I have made. In the course of 30 years, their number and variety have become formidable. Another source of information represents the mistakes that other pathologists have made in the performance of medicolegal autopsies. I have learned of these errors from reading their autopsy protocols or from performing second autopsies on exhumed bodies.

Inasmuch as I was not sure that I had either made or heard about all of the important mistakes that should be brought to your attention, I recently made inquiry of a group of colleagues who have had large experience in the field of forensic pathology. Their replies constitute my third source of information.

Emeritus Professor, Retired.

* Ward Burdick Award Address, presented at the Thirty-Fifth Annual Meeting of the American Society of Clinical Pathologists, Chicago, Illinois, October 11, 1956. Reproduced with permission from the American Journal of Clinical Pathology, Vol. 26, p. 1383, 1956.

VARIOUS MISTAKES IN FORENSIC PATHOLOGY

Mistake of not being aware of the objective of the medicolegal autopsy

I am sure that many, if not most, of the mistakes that are made stem from the fact that hospital pathologists are so often unaware of some of the important objectives of the medicolegal autopsy. It should be realized that the medicolegal autopsy is often expected to provide information that would not be looked for in an ordinary hospital case, i.e., information that is important for legal, rather than medical, reasons. An examination that would be entirely adequate by ordinary medical standards may be so inadequate from a medicolegal standpoint that a murder may not be recognized or an innocent person may be charged with a murder that was not committed. Thus, if the pathologist is to avoid mistakes in the performance of a medicolegal autopsy, and particularly in an instance in which homicide is a possibility, he should be aware that, in addition to determining the cause of death, he (and he alone) may have access to information that may be essential in establishing 1) the identity of the dead person; 2) the time of death; 3) the circumstances in which the fatal injury was sustained; 4) the type of weapon or agent that was responsible for the injury; 5) factors that may have predisposed the victim to injury, or modified the effects of the injury; 6) the identity of the person (or persons) responsible for the injury.

An excellent illustration of the importance of being aware of the objectives of the medicolegal

autopsy is provided by the following case. I am sure that the pathologists who participated will remember it with great embarrassment. The sudden and somewhat mysterious death of a notorious person in a tavern aroused a good deal of public interest, especially after the coroner had ruled, without benefit of autopsy, that death resulted from natural causes. Arrangements were made for an autopsy and two locally prominent hospital pathologists were engaged for the task. Their examination disclosed that death resulted from traumatic laceration of the liver and massive intraperitoneal hemorrhage. This finding led to the arrest of a suspect who was charged with having killed the decedent by striking and kicking him in the abdomen. At the trial some weeks later, the defendant's attorney, in maintaining his client's innocence, alleged 1) that the decendant had been drunk and quarrelsome at the time of the alleged assault, and that, as a result of this, he had made an unprovoked attack on the defendant, striking him repeatedly on the head and face with his fists, and 2) that the decedent had sustained his fatal injury by falling against the sharp corner of a table, rather than by being struck or kicked by the defendant. If these statements were true, the defendant was innocent. It was expected that the autopsy findings would throw some light on the true facts.

When the pathologists who performed the autopsy were on the witness stand, the attorney brought out the fact that they did not 1) test the dead man's blood or brain for alcohol, 2) examine his hands for evidence of bruises that would be consistent with his having been in a fist fight, or 3) examine his abdominal wall for injuries that might indicate the kind of impact that resulted in the ruptured viscus. The defendant was acquitted, but no one knows whether his story was true or false. If these pathologists had been better informed in regard to the objectives of the medicolegal autopsy, they almost surely could have obtained evidence that would have helped to establish whether the defendant's account of the altercation was true or false.

Mistake of performing an incomplete autopsy

A partial autopsy is always a mistake in a medicolegal case. The finding of coronary disease, presumably of sufficient extent to account for death, is the reason most often given for the premature termination of a medicolegal autopsy. Obviously, the existence of coronary disease, even though it is extensive, does not exclude the possibility that death resulted from injury or poisoning. Failure to perform a complete autopsy, or to save material for toxicologic analysis, is a dangerous practice in any instance of clinically unexplained death, even though it appears that an acceptable cause of death has already been demonstrated.

Many pathologists do not ordinarily examine the cervical segment of the spinal column, the larynx, or the laryngopharynx, even though the cause of death was not recognized elsewhere. In a medicolegal autopsy, examination of these regions should never be neglected. If such an examination is not performed, there is an excellent possibility that a second autopsy may reveal an unsuspected injury of the cervical cord, with or without fracture of the spine, an impacted foreign body in the glottis or larynx, or the presence of laryngeal or perilaryngeal injuries that are indicative of manual strangulation.

Mistake of permitting the body to be embalmed before performing a medicolegal autopsy

The mistake of permitting a body to be embalmed before autopsy may be as disastrous as the performance of an incomplete autopsy. Even though the embalmer does not use a trochar, embalming invariably results in a wide variety of artifacts that tend to destroy or obscure evidence. Some of these are mechanical and some are chemical. Not the least is the fact that the embalming fluid renders the blood and tissues unfit for many toxicologic tests.

Mistake of regarding a mutilated or decomposed body unsuitable for autopsy

If the identity of the dead person or the cause of death is in doubt, do not make the mistake of advising that an autopsy is not worthwhile, owing to the presence of putrefaction, mutilation, or damage by fire. No matter how putrid or fragmentary the remains, careful examination is almost invariably productive of information that bears on the identity of the dead person, and such examination frequently discloses previously unrecognized evidence that is pertinent to the cause and manner of death. I have a vivid recollection of the case of an extensively burned body that was found in the smoldering remains of a burned-out farm hourse. The legs, arms, anterior wall of the chest and abdomen, and the top of the head had been destroyed. The coroner released the remains for burial, without further examination, inasmuch as he thought they were not suitable for autopsy. Subsequently, the body was exhumed for further examination, owing to the fact that an insurance company was not satisfied that the remains were actually those of the missing occupant whom they had insured. Examination of the charred remains disclosed 1) that the kyphotic condition of the spine and the edentulous condition of the mouth

indicated that the body was not that of the missing insured person, and 2) that death had been caused by a bullet wound, not by the fire. Considering the ease with which external evidence of murder might be destroyed by a conflagration, the desirability of performing autopsies on burned bodies is obvious.

Frequently the reluctance of a pathologist to perform an autopsy on a decomposed body is due to the odor and vermin, rather than to his belief that the examination would not be productive of evidence. There is rarely any legitimate reason for haste in the performance of such an autopsy, and storage of a decomposed body for 24 hours at near-freezing temperature will invariably mitigate the odors and tend to immobilize the vermin.

Mistakes resulting from nonrecognition or misinterpretation of postmortem changes

Bloating and discoloration. Pathologists who are not experienced in the examination of decomposed bodies are likely to form erroneous conclusions in regard to the appearance of a body during life. Gaseous bloating causes swelling of the lips, nose, and eyelids, as well as protrusion of the eyes. These changes, together with the generalized darkening of the skin, often make the face of the dead person completely unrecognizable. Moreover, the distention of the chest and abdomen tend to lead to a false impression of obesity. On several occasions I have reviewed autopsy protocols in which a dead body, described as that of an obese exophthalmic Negro, was actually that of a slender white person.

Vesication. One of the common phenomena of putrefaction is the formation of fluid-filled blebs beneath the epidermis. Such blebs are sometimes confused with vesication that results from antemortem burning.

Purging. After 24 hours in a warm room, bloody fluid is frequently purged from the mouth and nose of a dead body. If death was preceded by the development of pulmonary edema, a liter or more of fluid may be discharged. The finding of a body lying in such a pool of fluid has, on several occasions to my knowledge, led to the erroneous assumption that death was caused by a massive hemorrhage.

Nonuniform decomposition. It should be remembered that putrefaction tends to be accelerated wherever the skin has been broken or blood has accumulated in the tissue. Therefore, any region of the body surface where putrefaction is disproportionately advanced should be examined with particular attention to the possibility that it represents a site of antemortem injury. It is a frequent mistake to pass over such a region lightly, inasmuch as it seems to be particularly unfit for examination.

Rupture of the esophagus or stomach. Occasionally, and for unexplained reasons, agonal or postmortem digestion of the wall of the stomach or esophagus occus so rapidly that the contents of the stomach are found free in the peritoneal or pleural cavity within a few hours after death. If the autopsy fails to disclose the true cause of death in such an instance, the death may be erroneously attributed to an antemortem injury.

Autolysis of the pancreas. As in the case of the esophagus and stomach, agonal or postmortem autolysis of the pancreas may be well advanced within a few hours. The process may be local, general, or multicentric, and the autolyzed tissue often becomes dark brown, suggestive of hemorrhage. I have known experienced pathologists to misinterpret such changes as antemortem hemorrhagic pancreatitis.

Abnormal distensibility of the rectum, vulva, and vagina. After rigor mortis has dissipated, these muscular canals become readily distensible. Thus, the easy admission of a larger instrument or more fingers than the canal would have readily admitted in life is sometimes erroneously construed as evidence of antemortem injury incident to rape or sodomy.

Heat fractures. Bodies that have been exposed to protracted and excessive heat after death frequently develop explosive fractures of the skull. Mistakes are sometimes made when such fractures are attributed to antemortem injury.

Thermal "hemorrhage." The same internal stresses that produce thermal fractures of the skull frequently result in massive extravasation of blood into the epidural space. Such extravasations may occur before sufficient pressure has developed to fracture the skull and, therefore, they may be observed independently of skull fracture. The possibility of misinterpreting such a change is obvious.

Thermal fat embolism. Droplets of sudanophilic fat are commonly observed in the pulmonary vessels of badly burned bodies. I do not know whether such droplets are carried to the lungs from the burned skin during the agonal period, or if they are formed after death by coalescence of the invisible droplets of fat in the blood. On the other hand, I am sure that they do occur independently of antemortem trauma. Do not make the mistake of confusing this kind of a thermally induced artifact with traumatically induced pulmonary fat embolism.

Mistake of failing to make an adequate examination and description of external abnormalities

In the usual hospital death from natural disease, the examination of the surface of the body is ordinarily

a relatively unimportant part of the autopsy. Rarely, if ever, is the hospital pathologist concerned with the condition of the dead person's clothing. In a medicolegal autopsy, however, the clothing and skin always deserve careful scrutiny and a detailed description of marks of damage or soiling. The clothing should be examined before it is removed from the body, and the skin should be examined before it is washed.

A pathologist may think that the condition of a dead person's clothing is a nonmedical matter to be dealt with by the police. If the condition of the clothing reveals facts that are pertinent to the circumstances or manner in which the injuries were received, its evaluation should be the responsibility of a medically trained person. Thus, I remember a fatal stabbing case in which the examination of the dead woman's clothing provided the critical evidence that was required for the conviction of the murderer. The stabbing was said to have occurred in the front seat of an automobile in which the victim and a male companion were riding. According to her companion, a strange man entered through a rear door while the car was stopped at a traffic light. The stranger was said to have leaned over the back of the front seat and to have stabbed the woman twice. He then jumped out of the car and escaped. From an examination of the wounds, it was not determined whether or not they could have been produced in this manner. On the other hand, when the location and course of the wounds were compared with the knife-holes in her clothing and in the covering of the seat upon which she was sitting, it was evident that they were not produced by the downward thrusts that she was supposed to have received. It was this evidence that was largely responsible for the conviction of her companion.

Similarly, in examining the wound in the scalp of a victim of a hit-and-run accident, the finding and preservation of a fleck of green paint, which might have been ignored by a pathologist who was not experienced in such cases, provided the critical evidence that was needed to exonerate an innocent driver and to convict the guilty one.

Although it would seem to be obvious that the location, dimensions, shape, depth, and special features of every wound should be described, such information is frequently inadequately recorded on protocols that are prepared by pathologists who perform only occasional medicolegal autopsies. In the protocol of a medicolegal autopsy, it is better to describe 10 findings that prove to be of no significance than to omit one that might be critical.

Recently, I examined the voluminous protocol of an autopsy that was performed on the body of a person who died of injuries sustained under unknown circumstances. The pathologist used approximately 2000 words in his description of normal tissues, as well as of such abnormalities as adhesions, healed tuberculosis, a Chiari's network, a double ureter, and an ovarian cyst. The entire description of the injuries from which the woman died consisted of something less than 50 words. The district attorney commented that, although the document might be interesting to another pathologist, the protocol did not provide him with any useful information that was not in his possession before the autopsy was performed.

Mistake of confusing the objective with the subjective sections of the protocol

It is as surprising as it is distressing to note how frequently pathologists include statements of opinion and interpretation in the part of the protocol that is supposed to be objective and factual. The purpose of a protocol is twofold. One is to record a sufficiently detailed, factual, and noninterpretive description of the observed conditions, in order that a competent reader may form his own opinions in regard to the significance of the changes described. The other is to interpret the significance of the changes that were observed and described. Thus, a region of dark blue discoloration of the dermis, in the center of the volar surface of the left forearm, may or may not be a bruise. To refer to it as a contusion in the descriptive part of the protocol is to substitute an interpretation for a description, and this is as unwarranted as it may be misleading. A solid dark red, partially occlusive coagulum of blood in the lumen of an artery may be a thrombus, but to call it such is an interpretation that should appear on the diagnosis sheet, and not in the descriptive text.

In reviewing the protocol of a medicolegal autopsy, I found the description of the external genitalia of a teenage girl to consist of the statement that "the external genitalia showed extensive injuries which had been incurred incident to assault and rape." Considering the facts that the girl's body was lying in a field for 10 days before it was examined by the pathologist and the various parts of her body had evidence of mutilation by animals, it would obviously have been much better if the pathologist had recorded exactly what he saw when he examined the body, rather than his interpretation of the significance of what he saw.

The mistake of not examining the body at the scene of the crime

Almost without exception, the various experienced forensic pathologists who contributed suggestions for the preparation of this review stressed the mis-

takes likely to occur if the pathologist does not observe the body at the scene, and prior to disturbance of the body or its immediate environment. In some localities and under some systems of law enforcement, that is not feasible, however desirable it might be. The circumstances often indicate that such a visit would probably be a waste of the pathologist's time.

In many instances of death by unexplained violence, it is a fact that appreciation of the full significance of the autopsy findings may depend on evidence that may be obtained only at the scene, and before the body has been moved. Not only may the evidence required for evaluation of the postmortem findings exist only at the place where the body was originally found, but its potential significance may be apparent only to a medically trained person. Thus, in view of the pathologist's knowledge that the fatal injury was immediately incapacitating, it may be apparent to him that someone other than the dead person must have moved the weapon, must have rearranged the bed clothing, or must have left a trail of blood on the floor. A great deal of this type of evidence may be preserved by adequate photography. Frequently, however, the evidence at the scene is of such a nature that it should be examined by the pathologist in its original state if it is to be correctly evaluated.

Mistake of substituting intuition for scientifically defensible interpretation

This brings me to one of the most dangerous mistakes in forensic pathology, and one that is particularly prevalent among experienced forensic pathologists who, for one reason or another, acquire a propensity for what might be called "categorical intuitive deduction." This Sherlock Holmes type of expert may see certain bruises in the skin of the neck and conclude without doubt that they were produced by the thumb and forefinger of the right hand of the stranger. He may see an excoriation of the anus and maintain unequivocally and without benefit of other elements of scientific proof that the assailant was a sodomist. He ignores the essential component for proof of the correctness of any such scientific deduction, namely, the nonoccurrence of such lesions or changes in control cases. Such a pathologist usually has the happy faculty of failing to remember the many similar bruises of necks that were known to have been produced by mechanisms other than pressure by the thumb and fingers. He fails to remember that many anal and rectal excoriations that were caused by injuries other than sodomy. Such a pathologist is a delight to newspaper reporters owing to the fact that he "makes good copy." He may be highly esteemed by the police and by the prosecuting attorney because he is an emphatic and impressive witness. His prestige, together with his exclusive access to the original evidence, places him in an exceedingly powerful position in the courtroom. Rarely can the defense attorneys find anyone with comparable experience to evaluate the postmortem findings. On the other hand, if they do, it seems obvious to the jury 1) that the outside expert was hired to say something that would help the accused, and 2) that the outside expert, unlike the state's witness, was handicapped by the fact that he did not see the evidence with his own eyes.

It is difficult to estimate how much harm is done by these people. I know of a man who was hanged largely on the weight of such uncritical evidence. The ordinary hospital pathologist is not accustomed to being so continuously unchallenged as to permit him to acquire a full-blown God complex of the kind that I am discussing. The hospital pathologist must be able to defend his interpretations against clinicians who also have a certain amount of information about the facts in issue. It is only the full-time forensic pathologist who is likely to become accustomed to having his opinions go virtually unchallenged. The stakes are too high to play hunches in forensic pathology.

Mistake of not making adequate photographs of the evidence

If a negative or positive postmortem finding is so important that it may make the difference between the freedom or imprisonment, or the life or death of someone, every attempt should be made to protect, preserve, and record it for others to see and evaluate. No pathologist should regard himself so infallible that he is willing to carry such responsibility alone, if he can share it with others.

In other words, this is an exhortation that pathologists should prepare photographic records of all of the critical evidence that can be photographed, and particularly of evidence that might otherwise be altered or lost. It is one thing for the pathologist to state that he remembers that the fracture of the skull had a peculiar contour that corresponds to that of the hammer found in the back of the automobile belonging to the accused. It is quite another thing for the pathologist to have recorded this fact photographically. I recall a protocol in which the hole in the back of the head of a man who died of a through-and-through bullet wound was designated as the wound of entrance. No photographs were made. The description of the injuries was too meager for anyone else to form an opinion as to which was the wound of entrance and which was the wound of

exit. The shooting was not witnessed. If the bullet entered from the back, the probability of murder was supported. Proof of guilt rested almost entirely on the undocumented opinion of the pathologist who was relatively inexperienced in interpreting injuries caused by gunshot.

Recently, in one of our southern states, a person who was unjustly convicted of murder in a shooting case was released as a result of the pathologist who performed the original autopsy having made photographic records of his findings. This pathologist had originally misinterpreted the evidence, and, at the time of the trial, his misinterpretation was an important factor in bringing about the conviction. Inasmuch as he realized that he might have made a mistake, the pathologist eventually submitted the photographs for interpretations by more experienced forensic pathologists. It was then clarified that one of the wounds had been originally misinterpreted. This led to the reopening of the case and the subsequent release of the accused.

Another reason to make photographs, particularly of the surface of the body is that they provide a record of things that may not have seemed sufficiently important to warrant description at the time of the autopsy. In deaths by criminal violence, the presence or absence of a wide variety of seemingly inconsequential changes may subsequently prove to be important.

Mistake of not exercising good judgment in the taking or handling of specimens for toxicologic examination

Mistakes in this field are so varied and are made so frequently that it is difficult to know where to begin. Each of the pathologists whom I consulted in regard to this problem provided me with a list of mistakes that he had observed, and the lists are by no means identical.

Unclean containers. Specimens are often placed in unclean containers. Too frequently the pathologist who goes to an undertaking establishment to perform a medicolegal autopsy fails to take with him a sufficient number of clean containers for toxicologic specimens. Every toxicologist has had the experience of being requested to analyze for alcohol a sample of blood that was sent to him in a container which reeked of embalming fluid.

Contamination of specimens. The use of unclean containers is by no means the only cause of contaminated specimens. Thus, samples for toxicologic analysis of the liver, the brain, or other organs are often cut with the same instruments, and on the same surface upon which the stomach was opened.

Obviously, the significance of a given concentration of a substance in the brain is different from the same concentration of that substance in the stomach. The significance of the determination of mercury in postmortem material is frequently vitiated by possible contamination with Zenker's fluid in the autopsy room.

An excellent example of contamination of toxicologic evidence in the autopsy room is illustrated in the instance of a woman who was thought to have died of poisoning, as a result of having had cyanide thrown in her face. It was admitted in court that the pathologist used the same knife to obtain samples of tissue for toxicologic analysis that had been used to cut through the presumably contaminated skin of the upper part of the thorax. Inasmuch as the test for cyanide in her blood was equivocal, the presence or absence of cyanide in the lungs became a matter of critical importance. In the opinion of the defense, the finding of cyanide in the lung was without significance because of the probability of contamination. This was one of the principal reasons for the acquittal of the accused.

Permitting blood or tissue to putrefy. Every pathologist is probably aware that putrefaction may produce substances that yield false-positive tests for certain compounds, and that it may destroy other substances that would have yielded significantly positive tests. Why then would a pathologist allow a sample of blood or tissue to remain in a warm place for hours, or even days, before sending the material to the analyst? I dare say that this will happen next week in a dozen communities in the United States.

Inadequate samples. Samples submitted for toxicologic analysis are frequently too small. It is by no means uncommon for the toxicologist to receive a 20-ml test tube of gastric contents or a 120-ml tonsil bottle containing liver, with a request that he "examine for poison."

Send as much as you can—up to 500 g—of any material that you wish to have analyzed. If you wish to know only the level of alcohol or carbon monoxide in the blood, the sample may be smaller, but, even then, 20 ml is much better than 5 ml.

Poorly selected samples. Pathologists often fail to recognize that, from a chemical standpoint, the lumen of the alimentary canal is part of the external environment of the body. If they did, they would never limit their toxicologic specimens to the contents of the stomach or intestine. The finding of a foreign chemical substance in the contents of the alimentary tract usually means that the substance was ingested, but this finding does not necessarily establish the fact that a significant amount of the

substance was absorbed. Neither does the failure to find a poison in the alimentary tract exclude the possibility that the decreased person was fatally poisoned.

There is a great variation in the sites in which poisons may accumulate in the body after they have been absorbed, and extensive sampling is always desirable if the identity of the agent is not known. Of course, the material submitted for analysis should include any vomitus present and any samples of food, beverage, or medicines that may have contained poison.

Unlabeled specimens. Several years ago we received an unlabeled cardboard container in which there were six unlabeled jars and bottles. The messenger who brought it said that Dr. X would call us in regard to the shipment. Two of the bottles contained blood, two contained gastric contents, and two contained a mixture of tissues.

Dr X subsequently called and said that he had sent the contents of the stomach, some blood, and certain tissues from a man whom he suspected of having died of homicidal poisoning with barbiturate. Inasmuch as the containers were not full, we asked why he had sent six rather than three. His explanation was that the messenger probably made a mistake and picked up three containers that belonged to another case, specimens that he decided not to send in for analysis. Fortunately, no barbiturate was found in any of the specimens, but, if it had been found, it might have been difficult, or even impossible, to establish which of the two persons had been poisoned.

Continuity of responsibility for protection of evidence. When an analytic result contributes to the proof that a crime was committed, or that the defendant is responsible for damages, the attorney for the defendant has the right and the obligation to try to establish whether or not the material tested came, *in fact*, from the place from which it was said to have come, and was, *in fact*, in virtually its original condition. Among other things, this means that some responsible person must be able to vouch for the specimen during every minute of the time that elapsed between its collection at the autopsy and its analysis in the laboratory.

The fewer the persons involved in this responsibility, the better is the chain of evidence. The specimens should pass from hand to hand, and they should never be left in a place where they could be tampered with or become altered.

The analyst must also be certain that there is no reasonable possibility that the tube upon which he made his final test could have been confused with some other tube during the analytic procedure. This also applies to sections of tissue for microscopic examination. There must be no reasonable possibility that the section of tissue with the critical microscopic changes could have been confused by the histology technician with a section of tissue that was derived from some other place or person.

Preservation of the excess material. Whenever possible, the analyst should retain samples of original evidence, in order that it may be examined by other experts if such examination is authorized by the court. Frequently, when the settlement of a legal issue depends upon the results of a laboratory test, it is requested that the original material be made available for reexamination by another toxicologist. Such a request may be legitimate, and the pathologist or toxicologist who finds that this can not be done, because the material was exhausted or destroyed in making the original examination, is often in an embarrassing and even indefensible position. The deep freeze is an indispensable part of the equipment in a labortory where the staff deals with medicolegal evidence.

Facts bearing on identity of the poison. Too often the toxicologist receives specimens with no information, but only a request that he test for poison. If the person was found dead and if no information is available regarding the duration or nature of the fatal seizure, the toxicologist should be so informed. If a dead woman was known to be the mistress of a photographer, or if she was an employee in a silver-plating factory, the toxicologist should be told. A blind toxicologic analysis is a tremendous undertaking, and many days are required for its completion. I am in complete sympathy with the analyst who puts the specimen away, and forgets it, when he is told only to "look for poison."

Mistake of permitting the value of the protocol to be jeopardized by minor errors

Unless a pathologist has the experience of having been on the witness stand when his autopsy protocol was examined, word for word and line by line, by counsel for whichever side of the case was damaged by the evidence, he will not fully appreciate the potential gravity of this kind of mistake.

Assume that you are on the stand and have testified that the decedent came to his death as a result of an intracranial hemorrhage that was caused by one or more blunt impacts against the head. Assume further that you testified that the circumscribed depressed fractures of the skull are not consistent with their having been produced by an impact against a flat cement sidewalk, but that they are consistent with having been produced by a beer bottle that was shown to you as State's Exhibit No. 142.

Counsel for the defense does not like this evidence

and seeks to weaken or destroy it. He asks, "How does it happen that the date of the autopsy as recorded on the protocol is June 15, when the evidence shows that the death did not occur until June 16?" You explain that this is a typographic error.

Counsel asks, "Who witnessed the autopsy?" After reading the names of the three persons listed on the protocol as witnesses, it develops that the first name of one is incorrect, and that there was a fourth witness whose name was not included. Thirty minutes later, after disclosure of still other errors, it develops that you failed to notice that the left eye was prosthetic, and that, although you described the appendix as having no abnormality, the man unquestionably had an appendectomy some 5 years previously.

None of these mistakes has any real bearing on the important issue in the case, but, by the time they have emphasized and an articulate lawyer has commented on them, the jury begins to wonder whether any part of your report is reliable. There should be no mistakes in the protocol of a medicolegal autopsy, even though they seem to be unimportant.

Miscellaneous mistakes

Although the general areas in which most of the mistakes that are commonly made in forensic pathology have been discussed in the preceding paragraphs, there are certain specific examples of errors that deserve emphasis.

Errors of omission in the collection of evidence required for identification.

1. Failure to make frontal, oblique, and profile photographs of the face.
2. Failure to have fingerprints made.
3. Failure to have a complete dental examination performed.

Errors of omission in the collection of evidence required for establishing the time of death.

1. Failure to record the rectal temperature of the body.
2. Failure to observe changes that may occur in the intensity and distribution of rigor mortis—before, during, and after autopsy.
3. Failure to observe the ingredients of the last meal and its location in the alimentary tract.

Errors of omission in the collection of evidence required for other medicolegal purposes.

1. Failure to collect specimens of blood or brain for determinations of the contents of alcohol and barbiturates.
2. Failure to determine the blood group of the dead person if death by violence was associated with external bleeding.
3. Failure to collect nail scrapings and samples of hair if there is a reasonable chance that death resulted from assault.
4. Failure to search for seminal fluid if there is a reasonable chance that the fatal injuries occurred incident to a sex crime.
5. Failure to examine clothing, skin, and the superficial portion of the bullet tract for a residue of powder, and the failure to collect samples of any residue for the purpose of chemical identification.
6. Failure to use x-rays for locating bullets or fragments of bullets if there is any doubt in regard to their presence and location.
7. Failure to protect bullets from defacement, such as is likely to occur if they are handled with metal instruments.
8. Failure to collect separate specimens of blood from the right and the left sides of the heart in instances in which bodies are recovered from water.
9. Failure to collect samples of fluid from the air passages and stomach in instances where bodies are found in water.
10. Failure to strip the dura mater from the calvaria and base of the skull. Many fractures of the skull have been missed because the pathologist did not expose the surface of the fractured bone.

Errors of omission that result in the production of undesirable artifacts or in the destruction of valid evidence.

1. Opening the skull before blood is permitted to drain from the superior vena cava. If the head is opened before the blood has drained from it, blood will almost invariably escape into the subdural or subarachnoidal space, and such an observation may then be interpreted as evidence of antemortem hemorrhage.
2. The use of a hammer and chisel for opening the skull. A hammer and chisel should never be used for this purpose in a medicolegal autopsy. Fractures produced by the chisel are frequently confused with antemortem injury.
3. Failure to open the thorax under water if one wishes to obtain evidence of pneumothorax.
4. Failure to tie the great vessels between sites of transection and the heart when air embolism is suspected.
5. Failure to open the right ventricle of the heart and the pulmonary artery *in situ* if pulmonary thromboembolism is suspected.
6. Failure to remove the uterus, vagina, and vulva *en masse* if rape or abortion is suspected.

Mistake of talking too soon, too much, or to the wrong people

Too soon. The performance of a medicolegal autopsy in an instance of known or suspected hom-

icide is almost invariably a dramatic event. The reporters, police, district attorney, and even your colleagues, may exert pressure to find out what you think before you have completed your investigation. It may be a temptation, and, in the case of the district attorney, it is often desirable to give impressions of the situation even though all of the facts are not in. *If you do so*, be sure to make it clear that the impressions are tentative and subject to change.

An illustration of the danger of releasing opinions prematurely is illustrated by the following case. Owing to the facts that the dead woman's body was still warm and there was no rigor, Dr. X, the pathologist, told the district attorney, who was eager to know the time of death, that the woman probably died early that morning. He provided this information before the autospy had actually been started. The district attorney immediately passed this information to the reporters. Shortly before the noon edition of the papers reached the streets, the pathologist realized that he had talked too soon. The degree of autolysis of the parenchymatous organs, together with the presence of intravascular hemolysis, indicated a considerably longer postmortem interval than he originally thought. The noon edition of the newspapers carried two items on the first page, i.e., the district attorney's news release to the effect that Dr. X had established that the woman was murdered early that morning, and a recent news bullet stating that the murderer had just confessed that he killed the woman during the evening of the preceding day. Approximately 12 hours after the murder, and 2 hours before the body was found, the murderer moved her body from the warm room where crime occurred to the cool basement where the police found the victim. The interior of the body was warm because it had been in a warm place during most of the postmortem period. There was no rigor because rigor had developed and regressed. If the murderer had not confessed, and if he had been able to establish an alibi for the time that the murder was supposed to have been committed, he might have used the pathologist's premature and incorrect guess to support his innocence.

Too much. Do not let your desire to be helpful or to play the role of Sherlock Holmes lead you into the mistake of saying more than the facts warrant. A well-known pathologist in this audience once made this mistake, but I am sure he will not mind my telling you about it. An ex-gangster, whom many people might have wished to murder, was found dead on the floor of the lavatory in his own tavern. He had been shot through the right temple. No gun was found. There were no powder marks on the skin. This led the pathologist to think that the range of fire was several feet or more. The decedent had a recently blackened left eye. This observation, together with the absence of the gun and the absence of powder marks, stimulated the pathologist to tell the police that this seemed to be a clear case of homicidal shooting. He concluded that the dead man was in a fight in which he suffered a "black eye" and during which he was shot to death.

This opinion was subsequently found to be entirely incorrect; it is still resented by the district attorney and the police, and it is still a matter of embarrassment to the pathologist. The pathologist was not aware that subcutaneous echymoses in the orbital region frequently occur with gunshot injuries of the head, particularly those that involve the anterior fossa. He did not know that the contact between muzzle and skin in a suicidal injury by gunshot may be so perfect that no powder is deposited on the surface of the skin. He did not know that the teenage boy who visited the lavatory and found the dead man had pocketed the gun with the intention of telling no one about it. Obviously, he did not know that the decedent had written a suicide note to an ex-wife and put it in the mail an hour or so before he shot himself. In short, the pathologist made deductions that were not warranted by the evidence. He talked too much.

To the wrong people. The only persons who are entitled to information derived from the results of your investigation are the coroner, the district attorney, and the police. Never provide this sort of information to reporters unless you do so at the direction of the coroner or the district attorney. Be careful what you say in the presence of assistants in the autopsy room or employees of the undertaker. Such leaks of information are often the basis for unfounded suspicion of innocent persons or for the creation of unnecessary obstacles to the investigation by the police.

CONCLUSION

The almost complete exclusion from this discussion of the mistakes that are made in the interpretation of evidence has been deliberate. If evidence has been properly gathered and preserved, a mistake in interpretation may always be corrected. If the facts required for a correct interpretation are not preserved, the mistake is irreversible. □

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Putting It All Together: The Logic Behind the Forensic Scientific Method and the Inferential Test

Thomas W. Young, MD

During the last several years, I have published several articles on this website regarding the application of inference to forensic analysis[1-4]. Although there are a few references to logic in these articles, there is no detailed treatment of the logical principles that support the methods described in them, nor is there any detailed explanation of how the methods utilized commonly by many forensic scientists and pathologists are logically fallacious. The purpose of this article is to provide an explanation of the formal logic behind what I have written previously.

I am not a professionally trained logician. Much of what I have learned has been through self-study. For this article, I utilized a logic textbook recently published on the internet[5] [hyperlink to: http://www.coursesmart.com/978-0-07-731591-7?gclid=CMe_s_XB2qUCFQlubAodiBBNlw]. For those interested in reading further, page numbers from this e-textbook appear in brackets next to several of the concepts. This allows the reader of this article easy reference to the textbook because both items can be open at the same time on the computer monitor.

Some Basic Concepts

Logic is "the study of methods for evaluating whether the ***premises*** of an ***argument*** adequately support its ***conclusion***" [p. 1]. An ***argument*** is "a set of statements where some of the statements are intended to support another" [p. 1]. A ***conclusion*** is "the claim to be supported," and the ***premises*** are "the statements offered in support" [p. 1]. A ***statement*** is "a declarative sentence that is either true or false" [p. 2]. Each of the issues in a forensic analysis can be set up as a series of statements in an argument, and each argument can be evaluated for ***validity*** and ***soundness*** if it is a ***deductive*** argument [pp. 3-7] or for ***strength*** and ***cogency*** if it is an ***inductive*** argument [pp. 50-56]. Both the Forensic Scientific Method and the Inferential Test for Expert Testimony are based on ***deductive logic***, meaning that the "premises are intended to *guarantee* the conclusion" [p. 3]. I will discuss this first. ***Inductive logic*** only intends "to make the conclusion *probable*, without guaranteeing it" [p. 3]. I will discuss this last.

Deductive Logic, the Forensic Scientific Method and the Inferential Test

First, a reminder of the Inferential Test for Expert Testimony[2]:

> One can be reasonably certain if witness accounts of the past are consistent or not consistent with physical evidence in the present, but one cannot reliably surmise past events from physical evidence unless there is only one plausible explanation for that evidence.

Note the first part of the Inferential Test:

One *can be reasonably certain* if...

If the conclusion of an analysis can be guaranteed, then one by definition is able to be certain (to know for sure, to conclude beyond doubt). Deductive inference guarantees that one *can* be *reasonably certain* (certain after the use of one's reasoning), providing that the argument is ***valid.*** A ***valid argument*** is "one in which it is necessary that, if the premises are true, then the conclusion is true" [p. 4]. One way of ensuring a valid argument is to utilize a ***valid argument form*** [p. 16]. Two famous valid argument forms are ***modus ponens*** [p. 15] and ***modus tollens*** [pp. 19, 20]. The use of these forms guarantees that the conclusion will be true if the premises are true. *Modus ponens* is:

If *p*, then *q*; *p*; therefore, *q*.

The italicized letters *p* and *q* are variables. Statements can replace each of these variables. For example:

> **Gunshot wound example #1:** If a person presses the muzzle of a gun against his or her head and pulls the trigger, then he or she will get a contact gunshot wound in the head; Fred Phelps pressed the muzzle of a gun against his head and pulled the trigger (his wife saw it happen); therefore, Fred Phelps should have a contact gunshot wound in the head (*p* = pressing the muzzle of a gun against his head and pulling the trigger; *q* = contact gunshot wound in the head).

The first part of *modus ponens* has a ***conditional statement***. A conditional statement—a statement utilizing "if" and "then"—has an ***antecedent*** (*p*) and a ***consequent*** (*q*) [p. 17]. *In a forensic analysis, the conditional statement is a scientific principle derived from the biological and physical sciences.* The items in the statement can be general:

> If a person enters a crime scene, then he or she may leave evidence at the crime scene or some of the crime scene may leave evidence on him or her (Locard exchange principle).

Or specific:

> If a person is infected by chicken pox as a child, then he or she may develop shingles as an adult.

In the second and third parts of *modus ponens* as applied to a forensic analysis, *p* is the witness (or *anamnestic*) evidence and *q* is the physical evidence related to the witness evidence. The scientific principle is applied to the particular case at hand, such as in the gunshot wound example above.

Now, consider the steps of the Forensic Scientific Method[1]:

1. Acquisition of primary witness and other anamnestic evidence

2. Anticipation of future questions
3. Acquisition of physical evidence
4. Comparison of consistency of alleged events (hypothesis) with physical findings, obtaining additional data as needed
5. Assessment only to a reasonable degree of scientific certainty, recognizing the limitations of science

Step 1 involves learning *p*. Step three involves learning *q*. Step 4 involves comparing *p* to *q*. The conditional that applies to the case—symbolized as $p \rightarrow q$—is by its nature a hypothetical statement [p. 18]. The hypothesis in a case requires knowledge of *p* and how it leads to *q*. The hypothesis does not come from assessing *q* without knowledge of *p*.

Now, consider *modus tollens*:

> If *p*, then *q*; not *q*; therefore, not *p*.
>
> **Gunshot wound example #2:** If a person presses the muzzle of a gun against his or her head and pulls the trigger, then he or she will get a contact gunshot wound in the head; Fred Phelps does not have a contact gunshot wound in the head; therefore, Fred Phelps did not press the muzzle of a gun against his head and pull the trigger (his wife's statement is false) (*p* = pressing the muzzle of a gun against his or her head and pulling the trigger; *q* = contact gunshot wound in the head).

The first half of the Inferential Test—

> One can be reasonably certain if witness accounts of the past are consistent or not consistent with physical evidence in the present...

—involves applying *modus ponens* and *modus tollens*, both valid argument forms of deductive logic. With *modus ponens*, the witness account is consistent with the physical evidence as long as the physical evidence is adequately explained by the witness accounts according to a scientific principle expressed as a conditional statement. With *modus tollens*, the witness accounts are not consistent with the physical evidence when the physical evidence denies the truthfulness of the witness accounts according to a scientific principle expressed as a conditional statement. As long as the premises are correct, the truthfulness of the conclusion from these two famous argument forms is guaranteed, making it a ***sound argument*** (a truthful argument) [pp. 8, 9].

One point has to be made very clear. The first half of the Inferential Test can guarantee if a witness account is consistent or inconsistent with the physical evidence, but *a scientist can never guarantee that a witness account is truthful.* Consider the following ***truth table for the material conditional*** [p. 305]:

p	q	$p \rightarrow q$
T	T	T
T	F	F
F	T	T
F	F	T

We will ignore the second line of the table (scientific principles for this discussion are considered "true"). Line 1 represents *modus ponens* and line 4 *modus tollens*. Now note line 3. Lines 1 and 3 indicate that p may be true or false when q is true.

Consider this next example involving a three-year-old girl:

> **Example with three-year-old girl:** A frantic father called 911 to report that his child became suddenly unresponsive. She had defecated in her panties, and while he had her in the bathtub to clean her up, she became suddenly unresponsive. The 911 telephone operator instructed him on how to perform cardiopulmonary resuscitation (CPR) because he had never performed CPR before. Ambulance personnel discovered the child to be in cardiac arrest. The child was later pronounced dead. At the house, panties with feces lay in the bathtub. An autopsy disclosed abrasions in the upper abdomen, tears in the liver, hemorrhages along the colon in the upper abdomen, and small-volume blood loss into the abdomen. No abnormalities of the heart or brain were discovered. Subsequently, contrary to the father's account, the little girl's slightly older brother said that the father had struck the little girl in the chest when he had discovered that she defecated in her panties. He later described in his own way the little girl's sudden unresponsiveness, the father taking the girl to the bathtub, and the CPR performed by the father.

The physical evidence in the home of fecal stained panties and the autopsy evidence of frantic CPR with improper hand placement support both the father's and the older brother's accounts, both consistent with a sudden unresponsiveness occurring in the child from cardiac arrest. Both accounts are not the same in every respect, however. One account indicates a sudden cardiac arrest from unknown causes. This does happen. The account of the slightly older brother indicates a possible sudden heart stoppage following a blow to the chest, also known as *commotio cordis*. The absence of a bruise in the chest does not exclude such a blow to the chest.

In other words, two different witness accounts are consistent with the same physical evidence. Which account is true and which is false? We do not know. We do know that such an event is capable of happening according to the truth table above, so we stop short of guaranteeing the truthfulness of any witness account.

The beginning of the second half of the Inferential Test—

...but one cannot reliably surmise past events from physical evidence...

—describes the abductive inference or "backward reasoning" referred to in several of my articles[3,4]. The reason why one cannot reliably surmise past events from physical evidence is because that commits a formal logical fallacy known as the ***fallacy of affirming the consequent*** [p. 36].

If *p, then q; q;* therefore, *p.*

This is a *modus ponens* look-alike, but instead of affirming the antecedent (*p*), the fallacy occurs when the consequent (*q*) is affirmed instead. This is an ***invalid argument form*** [p. 34]. Use of this invalid argument form renders an argument unsound, even if the premises are true [p. 10]. Unsound arguments due to an invalid argument form are not reliable for determining and preserving the truth [p. 5]. As such, they should never be allowed as sworn testimony from a scientific expert unless they are accompanied by terms of uncertainty[2].

There are two exceptions to affirming the consequent as an invalid argument. One exception involves the use of a ***tautology*** [p. 332]. A tautology is a statement that is necessarily true. It can be represented as follows:

If *p*, then *p*; *p*; therefore, *p*.

Example of tautology: If Fred Phelps is a bachelor, then he is an unmarried man; Fred Phelps is a bachelor; therefore, he is an unmarried man (*p* = bachelor or unmarried man--both equivalent terms).

The courts do not deal with tautologies.

The second exception is when *p* and *q* are related to each other through a ***biconditional statement*** [pp. 306, 307]. This is also known as the ***if and only if exception*** [not mentioned in the textbook].

The conditional statement

If *p*, then *q*

can also be restated as

q, if *p*

or

p only if *q* [pp. 18, 19],

so stating

p if and only if *q*

is another way of stating

If *p*, then *q* and if *q*, then *p* [p. 287].

So (using but not listing ***simplification*** [p. 346] and other intermediate steps)

If p, then *q* and if *q*, then *p; q*; therefore, *p.*

A biconditional statement is symbolized as $p \leftrightarrow q$.

Now for the final portion of the second half of the inferential test:

...unless there is only one plausible explanation for that evidence.

This is another way of stating *p* if and only if *q*, with *q* being the result of the only plausible explanation, *p* (and there being no other explanation).

Using the example for this "unless" exception from my article about the inferential test[2]:

> **Example from "An Inferential Test...":** A body would be "discovered in a wooded area with numerous stab wounds to vital organs and multiple devastating blunt force head injuries" if somebody killed that person (manner of death homicide), and somebody killed that person *only if* the body of that person is "discovered in a wooded area with numerous stab wounds to vital organs and multiple devastating blunt force head injuries" (manner of death homicide and no other manner of death); Mary Jones was found in such a condition; therefore, someone killed Mary Jones (*p* = somebody killed that person; *q* = a body "discovered in a wooded area with numerous stab wounds to vital organs and multiple devastating blunt force head injuries").

Unlike the conditional statement, the biconditional statement does not represent a concept from the biological or physical sciences to be applied in a forensic analysis. It represents instead a "smoking gun" form of evidence that does not require an explanation from a scientist. This is why I stated in my inferential test article that this form of evidence is best considered by the jury as an ultimate issue once all the evidence is presented. Jurors—not scientists—should be the ones to apply this exception[2].

In my first article[1], I stated that the standard scientific method uses ***falsification*** and the forensic scientific method uses ***verification*** for ***witness evidence*** and ***falsification*** for ***circumstantial evidence*** (circumstantial evidence in this article means indirect evidence without witnesses but inferred from corroborating physical evidence). In order to infer deductively that the defendant is guilty in a circumstantial evidence case, the prosecutor has to rely upon the biconditional statement, $p \leftrightarrow q$, the exception indicated in the latter portion of the Inferential Test.

Consider the following ***truth table for the material biconditional*** [p. 306].

p	q	$p \leftrightarrow q$
T	T	T
T	F	F
F	T	F
F	F	T

The biconditional statement, $p \leftrightarrow q$, can be ***falsified*** (demonstrated to be false) if the ***truth values*** ("T" or "F") of p and q differ from each other. Frequently, a prosecutor may argue a case based on circumstantial evidence by stating that there is no other plausible explanation other than his or hers for the physical evidence to be what it is. The falsification of that argument involves providing another plausible explanation. The theory, $p \leftrightarrow q$, as proposed by the prosecutor (with p being the prosecutor's explanation of what took place), may be mistaken or false (there is another overlooked explanation of the event) or q may be mistaken or false (an important item of physical evidence was overlooked or misinterpreted).

Falsifying a conditional statement ($p \rightarrow q$) is another matter. Because a conditional statement in a forensic analysis often represents a scientific concept, only scientists performing experiments that control the antecedent and the consequent are capable of falsifying such a conditional statement—not the courts. Although the courts are not equipped to falsify conditional scientific statements (even though they are equipped to falsify biconditional statements), an attorney could at least demonstrate that the testifying expert's understanding and explanation of the scientific concept is mistaken.

It is important to point out, however, that most published studies concerning past event topics such as "child abuse" do not rise to the standard of being scientific concepts. This is because such studies involve past events that are not completely controlled or even known. The vast majority of scientists have never seen "child abuse" actually occurring (i.e. have never witnessed p). As such, compilations of such "experience" are merely affirming the consequent over and over again. Rather than scientific concepts, position papers of scientific organizations regarding topics such as "child abuse" are simply expressing ***conventions*** (conventions are ideas agreed upon by most scientists within an organization that may or may not be based on the scientific method[6]).

Regarding ***witness evidence***, the strategy is ***verification***. Biological and physical scientists use falsification in the standard scientific method to eliminate hypotheses (demonstrate that $p \rightarrow q$ is "F" rather than "T") in order to prevent committing the ***fallacy of incomplete evidence*** [p. 506] (an inductive fallacy--see below). The strategy of biological and physical scientists is to maintain an open mind and not to assume that the hypothesis is correct and that everything about it is known. To avoid the same fallacy, forensic scientists must rely upon verification (demonstrate that p is "T" rather than "F") so that they will avoid committing an injustice. Even

though the application of *modus ponens* cannot guarantee the truthfulness of a witness account even if it is consistent with the physical evidence, the truthfulness of the account is still assumed because of the given dictum that one is considered innocent until proven guilty. Also, an eyewitness is also considered more of an authority for what happened than a scientist because the witness actually saw what happened and the scientist did not (an inductive ***argument from authority*** [p. 511]—this inductive argument and others will be described below). If a witness account is initially falsified through the use of *modus tollens*, the scientist and the police investigator return to the witness to clarify the account rather than to assume that the account was complete and the circumstances surrounding the account thoroughly understood.

Arguments from Inductive Logic

There is some disagreement among logicians and other authorities as to the definitions of induction and deduction[5-7]. One definition of induction is that it involves "the inference of a general law from particular instances"[6], while deduction is "the inference of particular instances by reference to a general law or principle"[6]. The textbook[5] distinguishes a deductive from an inductive argument as between what is guaranteed as truthful (deductive) and what is probable without a guarantee of truthfulness (inductive), without the need to indicate what is general and what is particular. The authors of the textbook further argue that making such a distinction between general and particular statements for induction and deduction is a mistake, and they demonstrate this with multiple examples [pp. 507, 508]. They also make no reference to abductive inference[8,9]. For the sake of the discussion to follow, I will accept the textbook definition of what is inductive and what is deductive. In the end, it really does not make any difference which way we define these terms; both definitions seem to work out with this analysis.

As mentioned previously, an inductive argument does not guarantee the conclusion but only intends to make the conclusion probable. An inductive argument cannot be valid, because only deductive arguments are valid. With an inductive argument, one can affirm the consequent—an invalid argument form—and still have a ***strong argument*** and not a ***weak argument*** [pp. 50, 51]. A strong argument—an argument that "is probable that, if the premises are true, then the conclusion is true"—becomes a ***cogent argument*** if the premises are true [p. 53].

The forensic scientist or pathologist has the legitimate ability—or perhaps, the permission—to reason backwards in certain circumstances as long as the scientist uses *terms of uncertainty*: probable, possible, might happen, could happen, frequently happen, infrequently happen, suspicious for, cannot rule out, often associated with[2]. Also, early in a case when the information comprising *p* and *q* is not known in detail, reasoning backwards as a *heuristic* (essentially "a rule of thumb") can be useful in developing leads because what is probable in a case may lead to more information and may help to rule in and rule out contingencies[3].

Still, the use of inductive argument in the form of abductive inference (backward reasoning) in the courtroom setting—even when expressed with terms of uncertainty—is dubious and frankly should not be allowed, particularly when witness accounts are available for comparison with physical evidence[2,4]; nevertheless, the courts currently allow the free use of abductive inference. Consequently, it would be useful to analyze such an inductive form of inference in the courtroom by using inductive logic.

Before we do this, we need to understand what is meant by ***probability*** when it comes to forensic analysis. Inductive inference is concerned after all with what is probable and not probable, so such a discussion is important.

The textbook discusses three theories of probability: the ***classical theory***, the ***relative frequency theory***, and the ***subjectivist theory*** [pp. 545 - 551]. The classical and the relative frequency theories cannot apply to any past-event analysis because the number of possible outcomes or the number of observed outcomes—the denominators in both equations—is unknown and not subject to mathematical determination or manipulation. The subjectivist theory—where "probability is nothing more than degrees of belief" [p. 549]—is the form of probability that applies here. In this situation, the expert is essentially stating what kind of odds he is willing to give that a particular statement is true or false. For example, when an expert states that he or she is "95% sure that the injuries are from child abuse" (a statement actually made by one expert in a court case), the expert is willing to give 19-to-1 odds for child abuse if his or her bet were placed in a casino or at a horse race. When a professional expresses the willingness to make such a bet at such stakes, he expresses great confidence in his opinion. Is such confidence warranted? Would a logical analysis support this kind of confidence?

I now offer two strong arguments. The conclusion of the first strong argument is:

> **If eyewitnesses offer accounts that are consistent with physical evidence, then those accounts are probably truthful.**

This argument is so strong that if one of the witnesses claimed that he or she did not commit a crime, he or she should never have been arrested in the first place.

Earlier, I demonstrated that with deductive logic it is not possible to state with certainty if witness accounts are truthful or not truthful but only if they are consistent or not consistent with the physical evidence. Inferring truthfulness to a witness account would essentially affirm the consequent; nevertheless, even though we cannot guarantee the truthfulness of the witness account, we can offer a strong argument for its probability.

We can use an ***argument from authority*** [pp. 511 - 513]. An eyewitness may not possess an advanced degree or training in a biological or physical science, but he or she possesses a special kind of knowledge due to circumstances. He or she is one of the few individuals who were present to observe the incident in question. Furthermore, the lack of knowledge in the sciences ironically but definitely substantiates his

or her reliability as a ***cognitive authority*** ("a person or group possessing a special fund of knowledge") [p. 511] for the particular circumstance in question. If a witness or multiple witnesses offer statements that are consistent with physical evidence, then that person or persons cannot rely on knowledge of science to form educated guesses. The consistency of the statements would instead probably come from their truthfulness.

We can use ***induction by enumeration*** [pp. 513, 514]. Eyewitnesses do not offer only one observation but many observations. Past events are complex, so the multiple observations are complex in their number and their order. Another way of stating this is that p does not consist of one item but multiple items in succession (p_1, p_2, p_3, etc.). A greater number of consistent observations allow a higher probability for truthfulness because it only takes one observation to be false to make p inconsistent with q [pp. 335, 336]. Furthermore, if multiple witnesses independent of each other offer multiple observations and all of these observations are consistent with the physical evidence, then this increases the strength of an already strong argument by enumeration. Another way to put this is to say that a large number of ***confirming instances*** (a confirming instance is an "instance in which an implication of a hypothesis is observed to be true") [p. 526] offered by witnesses increases the likelihood that their statements are truthful.

Finally, we can argue on the basis of ***explanatory power***. The authors of the textbook in their description of scientific reasoning state, "a hypothesis has explanatory power to the extent that known facts can be inferred from it" [p. 526]. If explanations inferred from statements provided by witnesses explain phenomena observed by scientists during an autopsy or other scientific procedure, this increases the likelihood of the truthfulness of the statements. On the other hand, it has been my experience that "theories" (essentially, hypotheses) offered by prosecutors and others who characteristically believe that all defendants lie possess poor explanatory power, not only in the scientific sense but also in the common sense. In other words, the "theory" is frequently far-fetched.

Now for the conclusion of the second strong argument:

> **If an expert offers abductive inferences as opinions "made to a reasonable degree of medical or scientific certainty or probability" on the witness stand, then such opinions are probably incorrect (not truthful).**

This argument is so strong that an expert who infers this way would serve the cause of justice better if he or she never testified.

A treatise recently published by the National Academies of Science regarding forensic science discusses the "self-correcting" nature of proper science[10]. ***Self-correction*** involves questioning results and correcting errors and doing so as a formal and regular practice. Such self-correction currently does not exist among scientists for issues brought before a court. Instead, many experts make positive assertions on the witness stand and appeals to their own authority to do so. Having done

this, they possess neither the interest nor the ability to determine if their own assertions are truthful or not. This is because such a witness who abductively infers with certainty has neither the knowledge of the limitations for what he or she is doing nor the capacity to consider carefully the accounts of witnesses who were present to see what happened (if they had such capacity, they would not have offered abductive inferences in the first place). In spite of their appeals to their own authority (or the prosecutor's appeals to their authority), they commit an ***ad verecundiam fallacy*** (appeal to unreliable authority) [pp. 182 - 183, 512]. No matter the experience of the expert, that experience is unreliable if it is not consistently, formally and rigorously tested by first-hand witness accounts.

On the other hand, an expert who acknowledges the limitations of his or her science, who knows how to compare witness statements to physical evidence in deductive fashion, and who knows better than to infer abductively on the witness stand has a great capacity to self-correct. Such individuals actually learn from their experience, so their experience is probably reliable for courtroom purposes.

Also, abductively-inferring experts commit a ***fallacy of incomplete evidence*** [pp. 506, 512]. This fallacy occurs both at the outset of a case and when a case goes to trial. Experts who abductively infer from the witness stand familiarize themselves with *q* but characteristically know little about *p* at the outset of a case, either unwittingly or by choice. This leads them to affirm the consequent consistently at the outset. Once further information and arguments are advanced regarding *p* (if such information or arguments are even advanced), there is little interest in changing initial impressions--perhaps for reasons of pride, arrogance or self-preservation. This leads to an unwillingness to acknowledge the information or even to evaluate it carefully with an open mind.

Finally, such abductive inferences frequently have ***poor explanatory power.*** There is a human tendency to oversimplify the complexity of past events. Oversimplified explanations reflect a limited capacity to explain all the events that came before in a way that makes sense. Consequently, there is reference to general, vague notions—including diagnoses of "child abuse" and how injuries occur only with "three-story falls" or "thirty-mile-per-hour car crashes"—without any detailed or consistent explanation of how witness accounts and other physical evidence match up with their opinions.

Conclusion

I recognize that many consider the kind of analysis offered here to be too technical—something apart from their usual area of interest or expertise. I offer it nonetheless as a basis to exclude certain forms of expert evidence from courtroom proceedings, perhaps in the form of Daubert or Frye hearings. It is my hope that attorneys will take advantage of this information to attack illogic from experts in the courtroom.

It is also my hope that scientists, attorneys and judges will take the time to familiarize themselves with this material. Too many false allegations have been offered in the name of science and too many people have suffered serious damage—even lifelong damage—as a result. It is time to put all of this to an end.

References

1. Young TW. Forensic Science and the Scientific Method. http://www.heartlandforensic.com/writing/forensic-science-and-the-scientific-method. February 13, 2008.

2. Young TW. An Inferential Test for Expert Testimony. http://www.heartlandforensic.com/writing/an-inferential-test-for-expert-testimony. April 2, 2009.

3. Young TW. Is Sherlock Holmes' "reasoning backwards" a reliable method for discovering truth? Analyses of four medicolegal cases. http://www.heartlandforensic.com/writing/is-sherlock-holmes-reasoning-backwards-a-reliable-method-for-discovering-truth. September 7, 2010.

4. Young TW. Attorneys and Judges, You Can Stop the Madness Now. http://www.heartlandforensic.com/writing/attorneys-and-judges-you-can-stop-the-madness-now. September 18, 2010.

5. Howard-Snyder F, Howard-Snyder D, Wasserman R. The Power of Logic, 4th ed. New York: McGraw-Hill Higher Education; 2009.

6. McKean E, ed. New Oxford American Dictionary, 2nd ed, 2005.

7. Popper KR. The Logic of Scientific Discovery, 2nd ed. New York: Harper and Row; 1968. pp. 27, 32.

8. Peirce CS. Illustrations of the Logic of Science. Sixth paper—Deduction, Induction, and Hypothesis. The Popular Science Monthly 1878;13:470-82.

9. Commens Peirce Dictionary: Abduction. In: Bergman M, Paavola S, eds. The Commens Dictionary of Peirce's Terms. http://www.helsinki.fi/science/commens/terms/abduction.html. 2003-. Accessed on December 14, 2010.

10. National Research Council. The Principles of Science and Interpreting Scientific Data. In: Strengthening Forensic Science in the United States: A Path Forward. Washington, DC: The National Academies Press; 2009. p. 125.

Brain (1984), **107**, 15-36

Good!

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DELAYED DETERIORATION FOLLOWING MILD HEAD INJURY IN CHILDREN

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SUMMARY

A series of 42 children is described who, following a seemingly minor or trivial head injury, developed neurological signs after a lucid or symptom-free period. This group constitutes 4.34 per cent of 967 consecutive patients aged 2 months to 17 years who were seen by members of the neurological staff during the years 1978-1981.

Only one patient had an intracranial haematoma. The majority of patients showed a benign transient syndrome consisting of either convulsive or nonconvulsive signs with a spontaneous and full recovery. There were, however, 3 deaths in this series, apparently due to severe and uncontrollable unilateral or diffuse brain swelling, demonstrating the malignant counterpart of this benign syndrome.

The theories seeking to explain these phenomena are reviewed. Special reference is made to the hypotheses of Bruce and his associates regarding brain swelling as a causative factor. It is considered that an adequate theory to explain the pathogenesis is still lacking.

It is concluded that the juvenile brain responds to cranial trauma in a manner different from the adult brain. This implies a different approach in policy to hospital admission.

INTRODUCTION

It is a well established fact that children with head injuries who deteriorate following a lucid interval usually do not have an expanding intracranial haematoma, unlike adults where secondary deterioration following trauma is a strong indication that such a haematoma is present (Walton and Brooks, 1897; Pickles, 1949; Lindenberg *et al.*, 1955; Galbraith and Smith, 1976; James, 1979; Bruce *et al.*, 1979, 1981*a*).

Secondary deterioration not due to intracranial haematoma has been attributed to acute cerebral oedema (Pickles, 1949; Biemond, 1970), convulsions (Small and Woolf, 1957; Livingston and Mahloudji, 1961; Grand, 1974; Reilly *et al.*, 1975; Rose *et al.*, 1977), spreading depression of Leão (Oka *et al.*, 1977), a migrainous mechanism (Haas *et al.*, 1975), a functional disturbance of the rostral brainstem (Todorow and Feller, 1982), concomitant viral meningoencephalitis (Peters *et al.*, 1978), complicating meningitis (Rose *et al.*, 1977) or to unknown causes (Plum and Posner, 1980), whereas recent reports deal with the syndrome of diffuse cerebral swelling, which is attributed to acute cerebral hyperaemia (Langfitt and Bruce, 1975; Bruce *et al.*, 1979, 1981*a*, *b*). The CT scans of children with this diffuse cerebral swelling show a diminution or obliteration of the cerebral ventricles and

perimesencephalic cisterns, whereas the hyperaemia is related to increased CT attenuation numbers of the deep frontal white matter (Zimmerman *et al.*, 1978).

The present study was undertaken in order to investigate retrospectively the incidence of lucid or symptom-free periods in a consecutive series of head-injured children, to describe the different clinical pictures presented by these patients and to correlate the clinical findings with the results of both EEG and CT studies.

PATIENTS AND METHODS

The University Hospital of Groningen has approximately 1100 beds and serves a population of 400000 for primary referrals and 2 million for secondary referrals.

In 1972 a Head Injury Research Study was set up in collaboration with neurosurgical centres in Rotterdam, Glasgow and Los Angeles (Jennett *et al.*, 1977). All injured patients in whom a head injury is suspected are seen by staff of the Neurology Department at the Accident and Emergency Clinic and assessed in a uniform way, using the Glasgow Coma Scale (Teasdale and Jennett, 1974). In order to study the occurrence of lucid or symptom-free periods in children after head injury, the records of all head-injured children (aged 0 to 17 yrs), seen during the years 1978–1981, were reviewed. During this period a total of 967 patients in this age group were seen as primary referrals, that is, not having previously been admitted to another hospital. The neurological assessment usually took place within 1 or 2 h after the accident, in all cases within 24 h.

Patients were included in the present series if, after a head injury not causing unconsciousness or only a very brief period of unconsciousness (not exceeding 5 min), this symptom-free period was followed by a subsequent deterioration of the level of consciousness or by the onset of focal neurological signs, or both. The clinical findings of these patients were compared with the EEGs, skull radiographs and CT scans if available. Where appropriate, attenuation numbers expressed as EMI units of the deep frontal white matter were obtained as described by Zimmerman *et al.* (1978). These attenuation values were compared with those of an appropriate control group of children. Average CT attenuation numbers of the deep frontal white matter in a series of control scans, as will be described later in this paper, ranged from 11.7 EMI units (SD 2.08) to 19.5 EMI units (SD 2.42).

RESULTS

Fig. 1 shows the characteristics of the group of 967 consecutive juvenile patients. Of these, 5 were either dead on arrival or died within a few minutes afterwards. Fig. 2 shows the age distribution of the remaining 962 patients. These patients have been classified according to the Abbreviated Injury Scale (AIS; Ommaya, 1979), which is summarized in Table 1. Because post-traumatic amnesia is usually difficult to assess in children (especially in the younger ones), only the estimated period of unconsciousness has been taken into account.

As fig. 1 shows, 5 patients with linear skull fractures were not admitted, usually because the head injury had occurred more than 12 h previously. Of all admitted patients, 22 died (5.7 per cent), death being attributable to primarily extracranial lesions in 4 cases.

In a total of 40 children a history of a lucid or symptom-free period was found following a head injury which had caused no immediate loss of consciousness. This group of patients meets the criteria of a 'trivial injury' according to Jennett (1962, 1975) (no amnesia, haematoma or skull fracture, or linear fracture only). Two cases

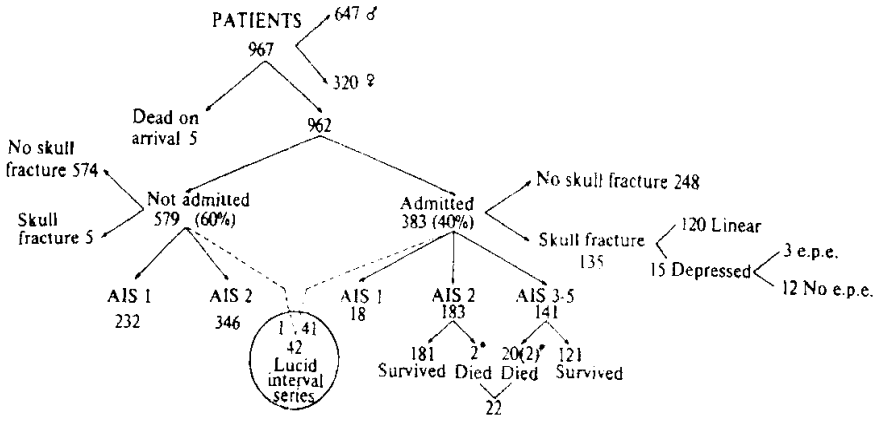

FIG. 1. Characteristics of the group of 967 consecutive patients, aged 2 months to 17 years. AIS = Abbreviated Injury Scale (*see text*). e.p.e. = early post-traumatic epilepsy. * = death attributable primarily to extracranial injuries.

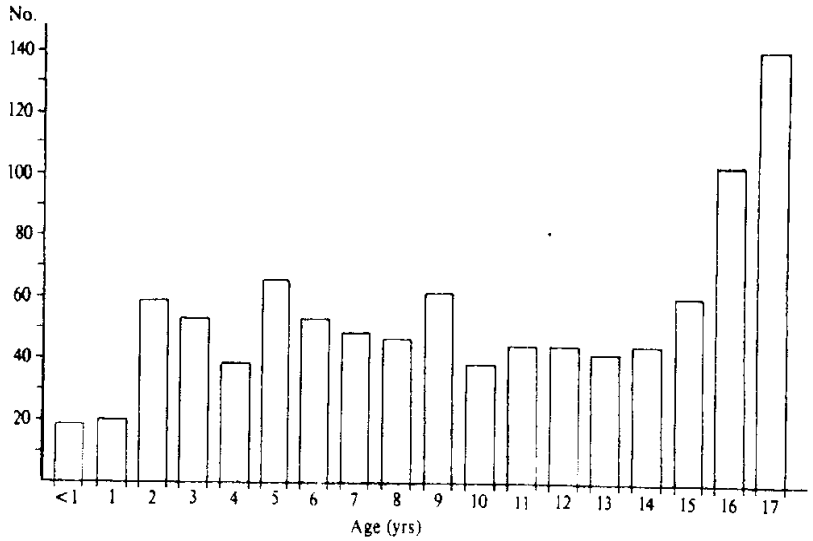

FIG. 2. Age distribution of 962 consecutive head-injured children, aged 2 months to 17 years.

TABLE 1. ABBREVIATED INJURY SCALE

Description of injury	*AIS code*	*Degree of injury*
Cerebral injury with headache; dizziness; no loss of consciousness.	1	Minor
Cerebral injury with or without skull fracture, with less than 15 min unconsciousness; undisplaced skull fracture; no post-traumatic amnesia.	2	Moderate
Cerebral injury with or without skull fracture, with unconsciousness more than 15 min, without severe neurological signs; brief PTA* (less than 3 h).	3	Severe (not life threatening)
Cerebral injury with or without skull fracture, with unconsciousness of more than 15 min with definite neurological signs; PTA 3–12 h. Compound skull fracture.	4	Severe (life threatening; survival probable)
Cerebral injury with or without skull fracture with unconsciousness of more than 24 h; PTA > 12 h; intracranial haemorrhage; signs of raised intracranial pressure.	5	Critical (survival uncertain)

* PTA = post-traumatic amnesia.

with a history of a brief loss of consciousness (not exceeding 5 min) followed by a lucid period, were included, bringing the total number to 42 (4.37 per cent of all patients). Forty of the patients were admitted immediately after the first neurological assessment, 2 had been allowed to go home, one of whom was subsequently admitted on the same day. In one patient the (short) transient period of deterioration had occurred several hours previously and at the time of neurological examination this patient had no symptoms; he therefore was allowed to go home. Fig. 3 shows the age distribution of the 42 patients. The patients were divided into two categories (fig. 4), the criterion being the occurrence or absence of convulsions (Oka *et al.*, 1977).

Group 1. Convulsive Cases

Thirteen children (31 per cent) had early post-traumatic seizures, occurring in the first week after injury (Jennett, 1962, 1969, 1975). Table 2 presents the characteristics of this group. Seven patients developed a focal or generalized status epilepticus, which necessitated intubation and controlled ventilation in 6. More than half of the patients developed these seizures within one hour after injury, 12 out of 13 within two hours, and only one after a longer period (28 h).

Case 1. A 5-year-old-boy fell off his bicycle. He did not lose consciousness and resumed cycling immediately. One hour after injury he developed focal seizures involving the right hand and arm. On admission one hour later, the boy was in generalized status epilepticus, which reacted promptly to intravenous diazepam. A CT scan performed within three hours after injury showed no abnormalities. The mean CT attenuation numbers of the deep frontal white matter were 15.9 EMI units (SD 2.59).

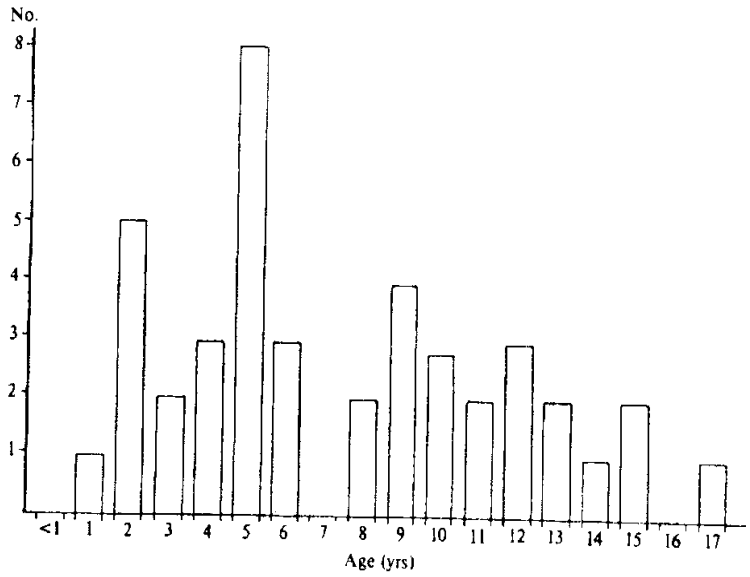

FIG. 3. Age distribution of 42 children with a lucid interval following mild head injury.

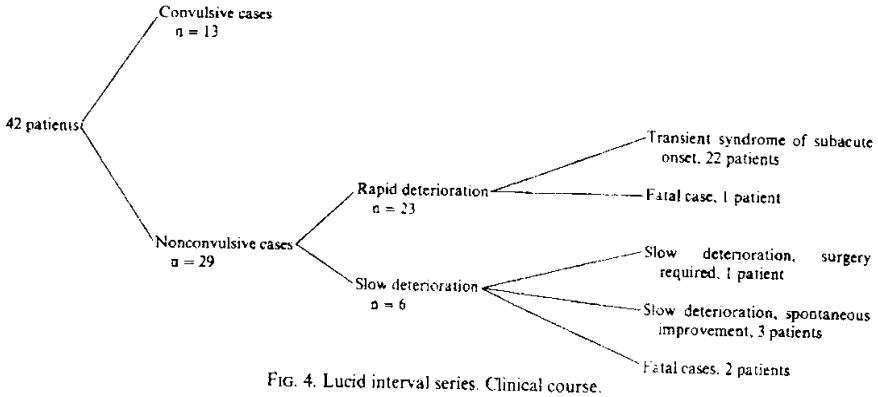

FIG. 4. Lucid interval series. Clinical course.

TABLE 2. CHARACTERISTICS OF CONVULSIVE CASES

Number: 13
Mean age: 7.3 years (range 1-17)
Cause of injury: fall, 10
traffic accident, 1
struck by object, 2
Skull fracture: 5 (38 per cent)
Onset after injury: within 1 h, 7 (54 per cent)
within 2 h, 12 (92 per cent)
Vomiting at onset: 3 (23 per cent)
Types of seizures: partial seizures, 4
partial + generalized seizures, 5
generalized seizures, 4
Status epilepticus: 7 (54 per cent)
Mean age of patients with status: 3.5 years (range 1-8)
Family history of epilepsy: 0
Past medical history: febrile convulsions, 1
rhesus incompatibility, 1
prematurity, 1
mental retardation, 1
early post-traumatic epilepsy, 1
Late epilepsy (follow-up 6 months to 3 years): 1

The ventricles were small, but the basal cisterns appeared normal. An EEG, performed within 24 h, showed asymmetry with localized slow activity in the left temporal region. There were multifocal isolated spikes and sharp-slow wave complexes, maximal in both temporal and parietal areas.

Several hours after admission the patient was fully alert, recalling all details of the injury. He was discharged after two days. Two EEGs, performed one and five months after injury, respectively, were normal. The past medical history of this patient revealed that he was premature and dysmature at birth. During the first fourteen days of life cyanosis had been seen on several occasions. Serial EEGs had not shown any abnormality at that time. He had been treated with anticonvulsants up to the age of five. Psychomotor development was normal.

The family history for epilepsy was negative in all patients. Five patients, however, had a past medical history (febrile convulsions, rhesus incompatibility, mental retardation or prematurity; 1 patient had had an identical episode of

TABLE 3. RELATION OF EEG ABNORMALITIES TO TYPE OF SEIZURE

	EEG					
Type of seizure	*N*	*D*	*F*	*D+F*	*E*	*F+E*
P	1		2	1		
P+G		1		1		2
G			1	1	1	

Type of seizure. P = partial seizure. PG = partial + generalized seizure. G = generalized seizure.
EEG. N = no abnormalities. D = diffuse abnormalities. F = focal abnormalities. E = epileptic discharges.

convulsions after a previous trivial injury). One patient possibly had a rubella infection and was readmitted with a second episode of status epilepticus a week later at which time an increased rubella titre was found. Apart from this patient no recurrence of seizures was noted in the follow-up period (six months to three years).

One or more EEGs were performed in 11 cases, usually within 24 h after injury (range 6 h to 9 days). Table 3 shows that there was no relation between the EEG findings and the type of seizure.

In only 2 patients was CT performed immediately following the convulsions. Both were normal. The densities measured in the deep frontal white matter were 15.9 EMI units, SD 2.59 (Case 1) and 11.3 EMI units, SD 1.60, respectively.

Group 2. Nonconvulsive Cases

These 29 children did not develop early post-traumatic seizures. In the majority (23 cases), there was a history of acute or subacute deterioration (i.e. from a level of no signs, the full clinical picture was reached within 15 min) at varying times after injury, whereas in the remaining 6 cases there was a slow, gradual deterioration.

Cases with rapid deterioration. A. Patients with a transient syndrome of acute or subacute onset. Patients were included in this group if the following criteria were met. (1) Acute or subacute onset. (2) Signs ending quickly, sometimes abruptly, mostly after a short period (not exceeding 12 h). (3) Nature of signs: loss of consciousness with or without focal neurological signs, focal neurological signs only or a period of severe restlessness or confusion following a period of normal behaviour.

This subgroup consists of 22 patients whose characteristics are listed in Table 4.

TABLE 4. CHARACTERISTICS OF NONCONVULSIVE CASES WITH A TRANSIENT SYNDROME OF ACUTE OR SUBACUTE ONSET

Number: 22
Mean age: 6 years (range 2–13)
Cause of injury: fall, 15
traffic accident, 5
struck by object, 2
Skull fracture: 5 (23 per cent)
Onset after injury: 5–30 min, 12
30–60 min, 4
1–2 h, 5
36 h, 1
Duration of signs: 15–30 min, 5
30 min–2 h, 2
2–12 h, 15
Vomiting at onset: 21 (95 per cent)
Signs: disturbances of sensory level only, 7
disturbances of sensory level with focal neurological signs, 9
focal neurological signs only, 1
confusion, 5

The past medical history was unremarkable in all but 3 cases. One girl (aged 12 years) was known to have behavioural problems and EEG abnormalities (bilateral synchronous spike and wave complexes) without history of seizures. One boy (aged 5 years) had had febrile convulsions in the past. There was a family history of epilepsy but no history of seizures in another boy (aged 6 years), whose EEG subsequently showed spike and wave complexes. One patient was admitted with fever, the CSF showing no abnormalities.

There were remarkable differences in the severity of the presenting clinical picture as is illustrated by the following case histories.

Case 2. A 2-year-old boy fell down some stairs. He cried immediately. One hour later he became unresponsive within a few minutes. On admission he was obtunded with a left hemiparesis. A CT scan, performed within three hours of injury, was normal, with frontal white matter attenuation values of 11.7 EMI units, SD 1.57. Within four hours after the injury the hemiparesis disappeared and the level of consciousness became normal. Two days later the boy was discharged. An EEG, performed on the same day, showed slow waves over the whole of the right hemisphere.

Case 3. A 5-year-old boy was struck by a car. He was unconscious for less than five minutes. In the ambulance the right pupil dilated while he was talking. He subsequently became comatose with bilaterally dilated unreactive pupils. On admission, one hour after the accident, he was awake with normally reacting pupils. The EEG (day 5) showed only slight diffuse abnormalities. He was discharged after six days.

A similar short-lasting but alarming picture, consisting of deep coma with bilaterally unreactive pupils, resembling acute tentorial herniation by a rapidly expanding mass, was seen in one other patient in this group, a girl aged 12 years. In this patient these signs occurred 36 h after injury. The CT scan performed at that time showed small ventricles with obliterated cisterns (fig. 5). The deep frontal white matter densities were 17.4 EMI units, SD 2.47.

The most frequently observed focal neurological signs were pupillary abnormalities (in 4 out of 10 patients), followed by conjugate eye deviation and hemiparesis. A combination of focal signs was seen in 2 children. Two patients presented with transient blindness. In both patients the onset of blindness (some minutes and one hour after injury, respectively) occurred in combination with sudden vomiting. The period of blindness lasted less than one hour in both cases and ended abruptly. One child had struck his forehead, the other had fallen on his occiput.

In this group of patients, 6 EEGs were performed within one to five days after injury. Three of these showed local slow activity, 1 slight diffuse abnormalities, whereas bilateral synchronous sharp and slow wave complexes were noted in 2. A CT scan was performed in only 2 cases (*presented above*).

B. *Fatal case with a rapid deterioration. Case 4.* An 8-year-old boy jumped from a slowly moving cart and fell on his head. He got up immediately, walked towards his father and said 'I feel so funny in my head'. He then became flaccid. His mother, a nurse, subsequently stated that she had noticed dilated pupils at the time. On admission one hour later the boy was shocked and respiration was shallow. He was unresponsive and both pupils were dilated and fixed. No bruises were found on the head and there was no skull fracture. Echoencephalography showed no midline shift. The boy was

intubated and ventilated. Soon after admission massive pulmonary oedema developed. It was felt that the situation was hopeless and no attempts were made at further investigation. He died within hours of admission. Permission for donor nephrectomy was granted by his parents; no attempts were made to obtain permission for autopsy.

Slow deterioration. A. *Slow deterioration, treated surgically. Case 5.* A 10-year-old boy struck his head against a wall while being tossed by his friends. He was briefly unconscious, probably less than one minute, but recovered quickly. On admission he opened his eyes on request, obeyed commands and was fully orientated. There was a small occipital fracture. He deteriorated slowly over the following day. Laboratory screening revealed a clotting disorder, which was found to be a variant of von Willebrand-Jürgens disease. A CT scan showed a hyperdense area in the posterior fossa, which on exploration proved to be an extradural haematoma. He made a slow but excellent recovery.

B. *Slow deterioration with gradual spontaneous improvement.* In 3 patients (aged 10, 12 and 15 years, respectively) the signs developed slowly over a period of time after a lucid interval and then gradually resolved. In 2 patients these symptoms consisted of slowly progressive confusion with focal abnormalities in both the EEG and CT scan, located in the frontal region in one patient and in the left temporal region in the other. In the third case (Case 6) the deterioration consisted of more severe confusion, with signs of uncal herniation.

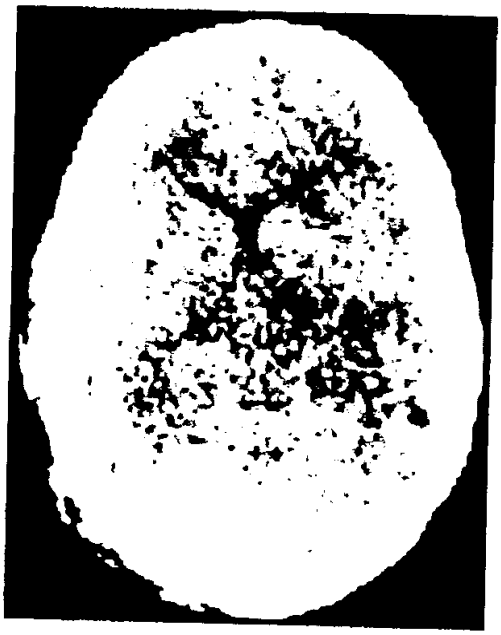

FIG. 5. Example of CT section at the level of the anterior horns and the mesencephalon. Note the narrow aspect of the ventricular horns which are not significantly displaced from the midline. The CSF cistern behind the quadrigeminal plate is obliterated.

Case 6. A boy aged 12 years was hit by a car. He did not lose consciousness. On admission he was lucid but nauseated. There was no skull fracture. Twelve hours after the accident he started to deteriorate with confusion and an enlarged pupil on the right. Angiography (because of unavailability of our CT scanner) showed a shift of the midline vessels with signs of a swollen right temporal lobe. No avascular mass was discovered. Several hours later his level of consciousness started to improve and the pupils became normal. The boy remained drowsy for two more days after which he recovered quickly. An EEG performed four days after injury showed diffuse slowing together with a local area of slow activity in the right temporal region.

C. *Fatal cases with slow deterioration. Case 7.* A 13-year-old girl fell off her bicycle. She did not lose consciousness at the time. On examination, three hours after the accident, she was lucid but nauseated. No neurological abnormalities were found. There was no skull fracture. She was sent home. Several hours later she developed a left hemiparesis and became drowsy. She was admitted to our hospital. A CT scan showed a marked shift of the compressed ventricles to the left. No abnormalities in the brain parenchyma were detected. Unfortunately raw CT data are no longer available, so no frontal white matter attenuation values can be measured in retrospect. An angiogram, performed in order to exclude an isodense haematoma, showed slowed intracerebral circulation, but no avascular mass. The girl was intubated and ventilated; high doses of corticosteroids were given, together with mannitol. She deteriorated over the next few hours and died on the following day. Autopsy was not performed.

Case 8. A boy aged 9 years fell off his skateboard. He resumed playing immediately, not having lost consciousness. Later that day he watched television for several hours and then went to bed. The next morning he complained of a headache and told his mother that he had difficulty in walking. Some time later he became stuporose and vomited. On admission, 16 hours after the accident, he did not open his eyes to painful stimuli, but he located these adequately while moaning. A bruise was noted in the right parietal area. There was no skull fracture. The left pupil was dilated, the right pupil somewhat smaller; neither reacted to light. While the patient was being examined both pupils started to react to light spontaneously. As the CT scanner was not available, an angiogram had been scheduled. It was felt, however, that the spontaneous improvement made an expanding intracranial mass less likely. Without having further deteriorated in the meantime, the boy suddenly developed fixed dilated pupils and apnoea. He was immediately taken to the operating theatre where bilateral burr holes revealed a swollen brain but no haematoma. The boy died 15 hours after admission. Consent for autopsy was refused.

DISCUSSION

The largest series of head-injured children, 4465 consecutively admitted cases, was reported by Hendrick *et al.* (1964). It was shown that almost 50 per cent of those who died were not in coma at the time of admission to hospital. Also, a history of a short or prolonged lucid period was present in 0.8 per cent of the patients, whereas the level of consciousness deteriorated gradually from the moment of impact in 1.8 per cent. Only the last category was correlated with a high percentage of intracranial, mostly extradural, haematomas.

In this study we assessed children who, following a minor or trivial injury, developed neurological signs following a lucid or symptom-free period. As these included cases with focal neurological signs in conscious children and early post-traumatic convulsions not leading to coma, it is not possible, strictly speaking, to use the term 'lucid interval', which refers to level of consciousness only, in these cases.

Therefore the criterion 'symptom-free' has been added. It does not seem illogical to use these criteria because in this way it is possible to focus attention on all those patients who demonstrated disturbed cerebral function after an injury which at first had appeared to be minor or even trivial.

In 4.37 per cent of 962 patients such a symptom-free period was encountered. In only one of these patients was an intracranial haematoma subsequently found. The remaining 41 patients showed a syndrome consisting of either convulsive or nonconvulsive signs.

Post-traumatic Epilepsy

Early post-traumatic epilepsy was noted in 16 head-injured children (1.6 per cent of our total series of 962). The 3 patients not having had a lucid interval all had a depressed fracture (fig. 1). According to Jennett (1962, 1973, 1975), early post-traumatic epilepsy is seen in about 5 per cent of all head-injured patients admitted to hospital. In children under 5 years of age, however, the incidence is twice as high. Hendrick and Harris (1968) also give an incidence of about 10 per cent in this age group. In patients under 5 years of age we found a percentage of 3.2 per cent. The difference between the incidence found by Jennett and by Hendrick and Harris, and by us, probably reflects the distinction between patient populations in neurosurgical and neurological departments.

According to Jennett, epilepsy rarely follows a trivial injury except in children under 5 years. The only adults encountered with epilepsy after trivial injury are those with 'immediate' epilepsy, when a seizure occurs at the moment the head sustains an impact. In the present series, the occurrence of early post-traumatic epilepsy was not limited to the under 5 age group, as 6 of the 13 patients were between 6 and 17 years of age. A similar age distribution has been described by Grand (1974).

Two explanations are possible for the relatively high frequency of early post-traumatic epilepsy following trauma in children as compared to adults (Jennett, 1962, 1975). The first is that children are predisposed to epilepsy, related to underlying but hitherto undetected brain damage. The second, favoured by Jennett and by Oka *et al.* (1977), is that children are somehow more liable to react to stress of different kinds by a seizure. Our finding that in almost half of the convulsive cases there was, albeit ill defined, a past medical history (*see* Table 2), which may have resulted in a liability to develop convulsions, gives some support to the first possibility expressed by Jennett. According to this author, the major significance of early post-traumatic epilepsy is the risk of late epilepsy, which he found to be 25 per cent. Focal early epilepsy in children was the only kind of early epilepsy in his series which did not significantly increase the risk of late epilepsy. Except for the child that was readmitted in a second episode of status epilepticus one week after the accident, no other cases of late epilepsy were encountered during the follow-up (three months to three years) of our patients with early epilepsy following trivial injury (Table 2). Oka *et al.* (1977) did not find a single case of late epilepsy in their series.

In the literature several explanations have been offered for the delayed deterioration in children following minor injuries. Several authors considered both convulsive and nonconvulsive signs as part of one syndrome and have attempted to give an explanation for its occurrence (Biemond, 1970; Haas *et al.*, 1975; Oka *et al.*, 1977); others have confined themselves to giving an explanation for the occurrence of convulsions only (Livingston and Mahloudji, 1961) or of nonconvulsive signs only (Walton and Brooks, 1897; Pickles, 1949; Gjerris and Mellemgaard, 1969; Bruce *et al.*, 1979, 1981*a*, *b*; Todorow and Feller, 1982).

Spreading Depression of Leão

By dividing our patients into two groups, convulsive and nonconvulsive cases, we follow Oka *et al.* (1977). They described a series of 37 children in whom transient neurological disorders occurred in the acute stage of trivial head injury and for whom no surgical treatment was subsequently required. The ages of their patients ranged from 10 months to 21 years, but the majority were under 14 years of age. Of these 37 children, 28 developed convulsive attacks and 9 children demonstrated nonconvulsive signs following a head injury without initial loss of consciousness. As in our series, most of the convulsive patients were under 8 years of age. Our group of patients with a transient syndrome of subacute or acute onset (Table 4) shows the greatest resemblance to their group of nonconvulsive cases, although our patients tended to be younger. They considered the nonconvulsive signs to be the primary and basic disturbance and they regarded the convulsive attacks as secondary phenomena. According to these authors, both phenomena can be explained as manifestations of the experimental phenomenon designated as spreading depression of Leão. In rabbits, Leão (1944) found that weak faradic or mechanical stimulation of the exposed cerebral cortex elicits a characteristic response, consisting of a marked enduring reduction of the spontaneous electrical activity of the cortex. Typical discharges of experimental epilepsy were noted in cortical regions when these were reached by a spreading wave of depression. Oka *et al.* suggested that the fact that the nonconvulsive disturbance was followed by convulsions in the younger children could be ascribed to a maturational factor, but that the primary process, the spreading depression, was identical in both groups. Thus, according to these authors, only the age factor determines whether the syndrome of transient neurological disorders, which include headache, nausea and vomiting, pallor, somnolence, irritability and restlessness, stupor, hemiparesis and aphasia, is followed by convulsive attacks. We noted some differences between our convulsive and nonconvulsive patients, however, which may indicate that the two phenomena are not as closely linked as these authors suggest. First there was, as stated earlier, a past medical history in almost half of the convulsive patients, which may have resulted in a liability to develop convulsions. Secondly, although a nonspecific sign such as vomiting was noted at the onset of the deterioration of almost all patients in the nonconvulsive group (Table 4), this sign was only rarely seen in patients with early post-traumatic convulsions (Table 2). We were unable to confirm the finding

of Oka *et al.* (1977) that the convulsive attacks did not develop directly from a lucid interval, but were nearly always preceded by nonconvulsive signs to a greater or lesser degree.

A Migrainous Mechanism

Haas *et al.* (1975), in a study of 25 patients, described 50 attacks consisting of transient neurological signs following mild head trauma; 40 of the attacks occurred in children under 14 years of age. All attacks developed after a latent interval, generally of one to ten minutes. Attacks were grouped into 4 clinical types: (1) hemiparesis; (2) somnolence, irritability and vomiting; (3) blindness and (4) brainstem signs. Two patients had an epileptic seizure. Five of the children later had spontaneous attacks which resembled their triggered attacks closely enough to be considered the same experience. The authors concluded that the temporary post-traumatic syndromes mentioned by them can be seen as diverse manifestations of a common underlying process which involves the cerebral vasculature, and that they resemble spontaneous classical migraine attacks in juveniles closely enough to postulate an underlying mechanism similar to migraine. Several authors (*see* Oka *et al.*, 1977; Shinohara *et al.*, 1979) regard this mechanism as analogous or identical with the mechanism underlying migraine.

A Functional Disturbance of the Rostral Brainstem

Todorow and Feller (1982) have recently described 49 children with mild post-traumatic stupor. This 'sleepy state', which lasted for 3 to 5 h, occurred in 9 per cent of all children admitted after a minor head injury. The lucid interval ranged from 15 min to 3 h. In almost one-third of the patients minor neurological signs were noted (pupillary abnormalities, pyramidal tract signs or ataxia), while EEG abnormalities were found in more than half. These EEG abnormalities mostly consisted of slight to moderate slowing of background frequency and, in many cases, persisted for a considerable time (from 5 days to 6 weeks) after the clinical signs had subsided. The authors postulate that both the secondary disturbance of consciousness and the EEG abnormalities can be explained by a functional disturbance of the rostral brain stem, probably related to the lability of arousal mechanisms in children.

In our series we have not regarded sleepiness in itself as a sign of secondary deterioration. Many children will fall asleep some time after the shock of an injury and the subsequent examination and treatment in an unfamiliar hospital setting. We cannot exclude the possibility, however, that the children in the Todorow and Feller series who became somnolent without developing focal neurological signs may represent the mildest form of the 'delayed deterioration syndrome'.

Direct Contusion of the Cerebral Cortex

Livingston and Mahloudji (1961) reported 4 patients between 2 and 4 years of age who developed convulsive seizures after a latent interval following mild head injury. They postulated that, since this picture has only been seen in young children at an

age when the cranium is rather malleable, deformation of the cranium at the moment of impact produces direct contusion of the cortex.

Focal or Generalized Brain Swelling, Due either to Rapid Oedema Formation or to Cerebral Hyperaemia

For almost a century there have been reports of clinical findings in head-injured children and adolescents strongly suggesting the presence of a rapidly expanding intracranial haematoma, but with these findings apparently being due to some other condition. The first case reports were published by Walton (1898) and Walton and Brooks (1897).

Pickles (1949) concluded that transient acute cortical oedema, with associated capillary anaemia, best explained the rapid and complete recovery without operation. Biemond (1970) also considered rapid oedema formation to be the cause of the transient cortical dysfunction accompanied by loss of consciousness. He differentiated between the more common benign transient syndrome and the rare fatal cases, which he saw only three times during his long career. These three children died after an exploratory craniotomy yielding negative results. Post-mortem examination showed diffuse cerebral swelling with microscopic findings of hyperaemia in the smaller arteries and precapillaries. He concluded that there was evidence both of brain swelling and brain oedema and he considered the brain swelling to be the primary event, probably attributable to diencephalic lesions.

Lindenberg *et al.* (1955) described the post-mortem findings of children dying from blunt head injuries. Diffuse cerebral swelling was the most consistent finding. They concluded that this post-traumatic brain swelling is markedly more common in children than in adults and that it may develop after a seemingly minor head trauma without subsequent loss of consciousness. Similar findings have been recorded by other pathologists (Adams and Graham, 1972; Adams, 1975), who also reported that they could not find distinct abnormalities on histological examination.

Until recently, post-traumatic brain swelling has always been attributed to brain oedema, but the crucial evidence for this cause is lacking. According to Miller and Corales (1981), direct measurements of water content of the brain have not been reported in patients with head injury. The concept of brain oedema causing diffuse cerebral swelling has now been replaced by the alternative explanation of increased cerebral blood volume. Bruce *et al.* (1979, 1981*a*, *b*) and Zimmerman *et al.* (1978) described the CT appearance of diffuse cerebral swelling, which they considered to be the commonest CT finding in head-injured children. This CT picture consists of obliteration or narrowing of the lateral and third ventricles and perimesencephalic cisterns, while cerebral attenuation values are higher than on follow-up scans. According to these authors, these attenuation numbers (measured in the deep frontal white matter) are also higher than the normal range defined from values measured in normal paediatric CT scans and certainly higher than would be expected if brain oedema were the cause of this cerebral swelling, as an increase in water content of the brain is linearly related to a decrease in attenuation numbers.

The finding of increased cerebral blood flow (CBF) in the patients who showed this CT pattern of diffuse cerebral swelling is taken to suggest a relationship between the increased attenuation at CT, and this increased CBF and the cerebral swelling is therefore ascribed to cerebral hyperaemia. The association of diffuse cerebral swelling and hyperaemia has also been reported by Obrist *et al.* (1979) from the same institution. Cerebral metabolism studies in their hyperaemic patients yielded a very low oxygen uptake and arteriovenous oxygen difference, indicating that the high blood flow is a true 'luxury perfusion'. The cause of the cerebral vasodilatation, which allegedly leads to cerebral swelling, is as yet unknown. Meyer *et al.* (1971) found in their animal model that the areas which caused increases in CBF when stimulated were located in the pontine and midbrain reticular formation, the thalamus and the hypothalamus. Raichle *et al.* (1978) assumed that locus coeruleus stimulation may change both CBF and cerebral capillary permeability, probably through central vascular aminergic pathways. Recently, Mies *et al.* (1981), using autoradiographic techniques, observed an increase in cerebral blood flow during spreading depression in animals. This finding thus appears to link the theories of Bruce *et al.* (1979, 1981*a*, *b*) with those of Oka *et al.* (1977). The increase in CBF found by Mies *et al.* (1981), however, was linked with increased metabolism, which means that apparently the increase in CBF during spreading depression cannot be interpreted as 'luxury perfusion'.

There are some unresolved problems relating both to the CT appearance of diffuse cerebral swelling and to the theory ascribing this swelling to vascular engorgement. Until clearly defined criteria for minimal ventricular size in normal children are set, caution is needed in interpreting the sign of 'small ventricles' (Snoek *et al.*, 1979). The perimesencephalic cisterns are normally quite evident in children, but the same caution is needed in attaching any value to an alleged 'compression or narrowing'; in our opinion only clear absence of these cisterns on the CT scan warrants a conclusion of cerebral swelling.

The unresolved problems in the theory ascribing the CT picture of diffuse cerebral swelling to vascular engorgement are threefold. The first is the reliability of cerebral attentuation coefficients determined by CT. We feel that relatively small changes in CT attenuation numbers, as described by Zimmerman *et al.* (1978), should be interpreted with circumspection. We have attempted to reproduce the normal range of values established by these authors (14.6 to 16.6 EMI units in the deep frontal white matter of 18 normal hemispheres). We determined average attenuation numbers in the same fashion in circular fields of 150 to 350 pixels each in the deep frontal white matter of 6 hemispheres of children aged 1 to 12 years, in whom no signs of cerebral swelling could be found at CT. All the examinations had been performed on the same scanner (EMI 5005) using a 160 × 160 matrix, 10 or 13 mm slice thickness. In some subjects a scan time of 70 s per slice was selected, in others 20 s. The same variable scanning parameters were used in the other subjects whose CT characteristics are mentioned elsewhere in this study. In our 6 subjects white matter CT attenuation numbers ranged from a maximum average of 19.5 EMI units

per measuring field (SD 2.42) to a minimum of 11.7 EMI units (SD 2.08). This is a much larger spread of normal values than is presented in the material of Zimmerman *et al.* (1978).

A number of factors may influence the assessment of tissue attenuation coefficients as expressed in CT numbers.

(1) *Scanner characteristics.* These vary from one manufacturer to another with regard to acquisition of data (scanner generation, presence or absence of a water bag), as well as the method of reconstruction. Extreme caution must be exercised in any comparison of different types of scanners often functioning under dissimilar operating conditions (Speller *et al.*, 1981). With regard to a single CT scanner, consistency of performance may be influenced by the state of maintenance (McCullough, 1977). The EMI 5005 whole body scanner mentioned in our study is serviced by contract with the manufacturer. It is also interesting to note that, as the linear attenuation coefficient of water decreases with increasing temperature, there is a difference in CT attenuation numbers of 2.5 to 3.0 EMI units between water at room temperature and water at body temperature. This should be taken into account in calibration of the scanner (Bydder and Kreel, 1979). Factors such as voxel size (determined by slice thickness and area represented by a pixel) may be expected to influence the statistical accuracy of the attenuation measurement. It is also our experience that in the EMI 5005 scanner CT attenuation numbers determined in a 20 s rapid scan are usually significantly higher than in a 70 s slow scan performed in the same region. We have no explanation for this discrepancy. Finally, changes in position of the head within the scanner ring can cause variations in CT numbers measured in the same cerebral region in serial examinations, as can changes in kilovoltage between examinations (Levi *et al.*, 1982).

(2) *Factors related to the patient.* The most important of these appears to be skull thickness. As the x-ray beam traverses tissue, its composition is altered. The lower-energy photons are filtered out, and while the x-ray beam as a whole is attenuated, the mean kinetic energy of the remaining photons is higher. This beam-hardening effect is especially marked in substances with a high linear attenuation coefficient such as bone, and when a large thickness of skull has to be traversed, this will influence attenuation measurement of skull contents (Di Chiro *et al.*, 1978). Shifts of some 7.5 EMI units have been noted between maximum and minimum skull thickness (Payne and Latchaw, 1978).

(3) *Partial volume effect.* Cerebral grey matter has a mean attenuation number some 3 EMI units higher than white matter (Weinstein *et al.*, 1977; Phelps *et al.*, 1975). If the field of measurement located in the frontal white matter should inadvertently contain grey matter, this will induce an upward bias in the mean CT attenuation value through a partial volume effect. This could happen if the field of measurement were located too near to the cerebral surface in the CT section, but also if a CT section were selected for measurement which contains in its basal portion some of the frontobasal grey matter.

The minor increase in CT attenuation number of 1.6 EMI units described by

Zimmerman *et al.* (1978) in their group of patients with post-traumatic brain swelling achieves statistical significance when serial measurements are performed in the same patient using the same CT scanner under identical operating conditions. We would like to warn, however, against attributing diagnostic significance to marginal variations in mean CT attenuation numbers measured in the first CT examination in an individual patient.

The second unresolved problem in relating the CT picture of diffuse cerebral swelling to hyperaemia is as follows. Although the increase of 1.6 EMI units in ET attenuation numbers mentioned above is relatively small in the context of other variable factors influencing the measurements, it represents a large shift if it is to be explained solely on the basis of increase in blood content. The blood concentration of cerebral white matter is reported to be 2.2 per cent and of grey matter 5.6 per cent (Ladurner, 1978). The CT number for blood is given as 27 EMI units (New and Scott, 1975), and for subfrontal white matter as 16.5 EMI units (Arimitsu *et al.*, 1977). In order to cause an increase of the value for white matter in the order of 1.6 EMI units, its blood content would have to increase very substantially. By the same token doubling the cerebral blood volume would result in an increase in total cerebral volume of only 3.5 per cent, and an increase of this amount of blood volume is hardly ever seen (Raichle, 1979).

The third unresolved problem is that the crucial measurement is cerebral blood *volume* rather than cerebral blood *flow*, as measured by Bruce *et al.* (Miller and Corales, 1981). Determination of cerebral blood volume (CBV) by emission tomography in 4 head-injured children belonging to the series of Bruce *et al.* (1981*a*) failed to demonstrate an increase in CBV in 3 of them (Kuhl *et al.*, 1980). These authors state that the CT pattern characteristic of diffuse brain swelling may occur in head-injured children without discernible increases in CBV and that this picture therefore cannot be explained by increases in CBV alone.

The clinical significance of the finding of a CT appearance suggesting diffuse cerebral swelling caused by cerebral hyperaemia is as yet unresolved. The crucial question is whether such a swelling (leading to severely increased intracranial pressure only in exceptional cases) is the *cause* of the deterioration in head-injured children or whether in most patients it is an epiphenomenon caused by the same processes which lead to the clinical deterioration. Cold and Jensen (1980) found that hyperaemia following head injury is a common phenomenon in children, that this hyperaemic phase lasts for some days to several weeks and that it is of no prognostic importance. The CT pattern of diffuse cerebral swelling is seen in 29 per cent of head-injured children and adolescents. There is a higher incidence in patients with a low score on the Glasgow Coma Score (41 per cent with a score of 8 or less) than in patients with a score greater than 8 (15 per cent) (Bruce *et al*, 1981*a*). These authors do not specifically mention the rate of occurrence of this CT pattern in children with a lucid interval. Conversely, in the clinical description of their group of 63 juvenile patients with the CT pattern of diffuse cerebral swelling, 37 per cent had experienced some form of lucid interval. Within the latter group two subgroups are described. In

a typical case of the first subgroup (8 cases) there was a lucid interval with a period of talking and complete consciousness from minutes to hours after injury, followed by the onset of vomiting, headache and frequently pallor and sweating, associated with decreased alertness. These patients exhibited evidence of decreased spontaneous motor activity, and loss of spontaneous speech and eye opening. No mention is made of focal neurological signs. None of these patients progressed to coma according to the criteria of Teasdale and Jennett (1974) and the intracranial pressure when measured in these patients remained normal. All these patients made a good recovery. The other subgroup consisted of 15 patients who were unconscious following trauma and then had an interval associated with recovery of eye opening, occasional words and more spontaneous motor function. This was followed by rapid deterioration leading to coma (Glasgow Coma Score of less than 8). One of these patients died as a result of delayed brain swelling. The others all made a good recovery. The patients of our series are by definition only comparable with their first group of patients. As most of our patients showed a benign, often shortlasting syndrome, few CT scans were made. In only one out of 4 scans, performed in children with a rapid deterioration consisting of either convulsive or nonconvulsive signs, was the typical CT appearance consistent with diffuse cerebral swelling seen (fig. 5), which leads us to the conclusion that in the majority of cases deterioration apparently is not associated with such cerebral swelling and that, therefore, there must be other explanations for this rapid deterioration.

The CT scan of Case 7, a girl with a slow deterioration, was consistent with swelling of one hemisphere, a finding which has been described in CT scans performed in the period immediately after injury (Kobrine *et al.*, 1977; Waga *et al.*, 1979). In both cases this swelling was, on the basis of attenuation measurements, also attributed to vasodilatation and not to oedema. The results of the angiogram in our Case 6, also a child with a slow deterioration, support the interpretation that local damage can be followed by local swelling, associated with clinical deterioration. The clinical course, radiological and (in one case) surgical findings in our fatal cases, although not confirmed by autopsy, strongly suggest that 'malignant brain swelling' following trivial head injury in children does exist. Excluding the rare occurrence of intracranial haematoma, we feel, however, that the syndrome of delayed deterioration following mild head injury in children in fact covers a variety of pathological states and that neither the clinical nor the CT findings support the view that brain swelling caused by cerebral hyperaemia is the only or even the usual underlying cause for such deterioration.

We conclude that trivial injuries in children are not infrequently followed by a deterioration which is preceded by a lucid or symptom-free period and which is usually transient. It is very likely that only in a minority of affected children signs of such severity occur that these children will come under medical care. On clinical grounds it is possible to differentiate between several distinct pictures. At this moment it can only be speculated whether or not the different clinical pictures presented by these patients reflect different pathophysiological entities. With the

exclusion of the relatively rare extracerebral haematoma, these clinical pictures point either to focal or generalized cerebral dysfunction, both with and without seizures. Although the recent theories developed by Bruce *et al.* relate the occurrence of cerebral dysfunction with diffuse cerebral swelling to hyperaemia, insufficient data are available so far to discriminate between this swelling being a causative factor for the deterioration or just an epiphenomenon in the majority of cases. We feel that caution is needed in interpreting the findings of marginally increased attenuation values in CT scans with small ventricles and compressed perimesencephalic cisterns as a state of diffuse cerebral hyperaemia and also in basing a therapeutic regimen solely on this interpretation. The rarity of this CT pattern in our patients does not give support to the assumption that in the majority of children diffuse cerebral swelling was the cause of the transient deterioration. There is now some evidence, however, that for unknown reasons some patients develop severe and often uncontrollable unilateral or diffuse brain swelling, as indicated by the radiological and surgical evidence in 2 out of our 3 fatal cases.

The fact that not infrequently deterioration following a trivial head injury may occur in children makes it mandatory in our opinion to accept a rather strict admission policy for head-injured children, especially if there is an indication in the past medical history that cerebral damage has been suffered in earlier life. In a deteriorating head-injured child that was symptomless before, the possibility of an expanding intracranial mass will always have to be excluded, preferably by CT scanning, although few haematomas will be found. The remaining patients usually show a benign and shortlasting syndrome followed by a spontaneous and full recovery in most, but not in all cases.

REFERENCES

ADAMS J H (1975) Neuropathology of head injuries. In: *Handbook of Clinical Neurology*. Edited by P. J. Vinken and G. W. Bruyn. Amsterdam: North Holland. Volume 23, pp. 35-65.

ADAMS J H, GRAHAM D I (1972) The pathology of blunt head injuries. In: *Scientific Foundations of Neurology*. Edited by M. Critchley, J. L. O'Leary and B. Jennett. London: Heinemann. pp. 478-491.

ARIMITSU T, DI CHIRO G, BROOKS R A, SMITH P B (1977) White-gray matter differentiation in computed tomography. *Journal of Computer Assisted Tomography*, **1**, 437-442.

BIEMOND A (1970) *Brain Diseases*. Amsterdam: Elsevier, pp. 771-785.

BRUCE D A, RAPHAELY R C, GOLDBERG A I, ZIMMERMAN R A, BILANIUK L T, SCHUT L, KUHL D E (1979) Pathophysiology, treatment and outcome following severe head injury in children. *Child's Brain*, **5**, 174-191.

BRUCE D A, ALAVI A, BILANIUK L T, DOLINSKAS C, OBRIST W, UZZELL B (1981*a*) Diffuse cerebral swelling following head injuries in children: the syndrome of 'malignant brain edema'. *Journal of Neurosurgery*, **54**, 170-178.

BRUCE D A, SUTTON L N, SCHUT L (1981*b*) Acute brain swelling and cerebral edema in children. In: *Brain Edema*. Edited by M. de Vlieger, S. A. de Lange and J. W. F. Beks. New York: John Wiley, pp. 125-145.

BYDDER G M, KREEL L (1979) The temperature dependence of computed tomography attenuation values. *Journal of Computer Assisted Tomography*, **3**, 506-510.

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COLD G E, JENSEN F T (1980) Cerebral blood flow in the acute phase after head injury. I. Correlation to age of the patients, clinical outcome and localisation of the injured region. *Acta Anaesthetica Scandinavica*, **24,** 245-251.

DI CHIRO G, BROOKS R A, DUBAL L, CHEW E (1978) The apical artifact: elevated attenuation values toward the apex of the skull. *Journal of Computer Assisted Tomography*, **2,** 65-70.

GALBRAITH S, SMITH J (1976) Acute traumatic intracranial haematoma without skull fracture. *Lancet*, **i,** 501-502.

GJERRIS F, MELLEMGAARD L (1969) Transitory cortical blindness in head injury. *Acta Neurologica Scandinavica*, **45,** 623-631.

GRAND W (1974) The significance of post-traumatic status epilepticus in childhood. *Journal of Neurology, Neurosurgery and Psychiatry*, **37,** 178-180.

HAAS D C, PINEDA G S, LOURIE H (1975) Juvenile head trauma syndromes and their relationship to migraine. *Archives of Neurology, Chicago*, **32,** 727-730.

HENDRICK E B, HARRIS L (1968) Post-traumatic epilepsy in children. *Journal of Trauma*, **8,** 547-556.

HENDRICK E B, HARWOOD-HASH D C F, HUDSON A R (1964) Head injuries in children: a survey of 4465 consecutive cases at the Hospital for Sick Children, Toronto, Canada. In: *Clinical Neurosurgery. Proceedings of the Congress of Neurological Surgeons*. Baltimore: Williams and Wilkins, pp. 46-65.

JAMES H E (1979) Analysis of therapeutic modalities for head-injured children. *Child's Brain*, **5,** 263-271.

JENNETT W B (1962, 1975) *Epilepsy after Non-missile Head Injuries*. London: Heinemann. First edition 1962; second edition 1975.

JENNETT W B (1969) Early traumatic epilepsy. Definition and identity. *Lancet*, **i,** 1023-1025.

JENNETT W B (1973) Trauma as a cause of epilepsy in childhood. *Developmental Medicine and Child Neurology*, **15,** 56-62.

JENNETT W B, TEASDALE G, GALBRAITH S, PICKARD J, GRANT H, BRAAKMAN R, AVEZAAT C, MAAS A, MINDERHOUD J, VECHT C J, HEIDEN J, SMALL R, CATON W, KURZE T (1977) Severe head injuries in three countries. *Journal of Neurology, Neurosurgery and Psychiatry*, **40,** 291-298.

KOBRINE A I, TIMMINS E, RAJJOUB R K, RIZZOLI H V, DAVIS D O (1977) Demonstration of massive traumatic brain swelling within 20 minutes after injury. Case report. *Journal of Neurosurgery*, **46,** 256-258.

KUHL D E, LAVI A, HOFFMAN E J, PHELPS M E, ZIMMERMAN R A, OBRIST W D, BRUCE D A, GREENBERG J H, UZZELL B (1980) Local cerebral blood volume in head-injured patients. Determination by emission computed tomography of ^{99m}Tc-labeled red cells. *Journal of Neurosurgery*, **52,** 309-320.

LADURNER G (1978) Die Bestimmung des zerebralen Blutvolumens mit der Computer-tomographie in grauer und weisser Substanz. *Fortschritte der Neurologie-Psychiatrie und ihrer Grenzgebiete*, **46,** 369-381.

LANGFITT T W, BRUCE D A (1975) Microcirculation and brain edema in head injury. In: *Handbook of Clinical Neurology*. Edited by P. J. Vinken and G. W. Bruyn. Amsterdam: North Holland, Volume 23, pp. 133-161.

LEÃO A A P (1944) Spreading depression of activity in cerebral cortex. *Journal of Neurophysiology*, **7,** 359-390.

LEVI C, GRAY J E, MCCULLOUGH E C, HATTERY R R (1982) The unreliability of CT numbers as absolute values. *American Journal of Roentgenology*, **139,** 443-447.

LINDENBERG R, FISHER R S, DURLACHER S H, LOVITT W V JR, FREYTAG E (1955) The pathology of the brain in blunt head injuries of infants and children. In: *Proceedings of the Second International Congress of Neuropathology*. Part II. Amsterdam: Excerpta Medica, pp. 477-479.

LIVINGSTON K E, MAHLOUDJI M (1961) Delayed focal convulsive seizures after head injury in infants and children. A syndrome that may mimic extradural hematoma. *Neurology, Minneapolis*, **11,** 1017-1020.

McCULLOUGH E C (1977) Factors affecting the use of quantitative information from a CT scanner. *Radiology*, **124,** 99-107.

MEYER J S, TERAURA T, SAKOMOTO K, KONDO A (1971) Central neurogenic control of cerebral blood flow. *Neurology, Minneapolis*, **21,** 247-262.

MIES G, NIEBUHR I, HOSSMANN K-A (1981) Simultaneous measurement of blood flow and glucose metabolism by autoradiographic techniques. *Stroke*, **12,** 581-588.

MILLER J D, CORALES R L (1981) Brain edema as a result of head injury: fact or fallacy? In: *Brain Edema*. Edited by M. de Vlieger, S. A. de Lange and J. W. F. Beks. New York: John Wiley, pp. 99-115.

NEW P F J, SCOTT W R (1975) *Computed Tomography of the Brain and Orbit (EMI Scanning)*. Baltimore: Williams and Wilkins, pp. 263-267.

OBRIST W D, GENNARELLI T A, SEGAWA H, DOLINSKAS C A, LANGFITT T W (1979) Relation of cerebral blood flow to neurological status and outcome in head-injured patients. *Journal of Neurosurgery*, **51,** 292-300.

OKA H, KAKO M, MATSUSHIMA M, ANDO K (1977) Traumatic spreading depression syndrome. Review of a particular type of head injury in 37 patients. *Brain*, **100,** 287-298.

OMMAYA K (1979) Indices of neural trauma: an overview of the present status. In: *Neural Trauma*. Edited by A. J. Popp, R. S. Bourke, L. R. Nelson, and H. K. Kimelberg. New York: Raven Press, pp. 205-216.

PAYNE J T, LATCHAW R (1978) Variation and nonuniform CT number response for intracranial contents as a function of skull thickness and head size. *Journal of Computer Assisted Tomography*, **2,** 509.

PETERS A C B, VERSTEEG J, LINDEMAN J, BOTS G TH A M (1978) Viral meningoencephalitis and head injury. *Acta Neurologica Scandinavica*, **57,** 77-87.

PHELPS M E, HOFFMAN E J, TER-POGOSSIAN M M (1975) Attenuation coefficients at various body tissues, fluids, and lesions at photon energies of 18 to 136 keV. *Radiology*, **117,** 573-583.

PICKLES W (1949) Acute focal edema of the brain in children with head injuries. *New England Journal of Medicine*, **240,** 92-95.

PLUM F, POSNER J B (1980) Supratentorial lesions causing coma. In: *The Diagnosis of Stupor and Coma*. Philadelphia: F. A. Davis. Third edition, pp. 87-151.

RAICHLE M E (1979) Discussion. In: *Neural Trauma*. Edited by A. J. Popp, R. S. Bourke, L. R. Nelson and H. K. Kimelberg. New York: Raven Press. p. 49.

RAICHLE M E, GRUBB R L JR, PHELPS M E, GADO M H, CARONNA J J (1978) Cerebral hemodynamics and metabolism in pseudotumor cerebri. *Annals of Neurology*, **4,** 104-111.

REILLY P L, GRAHAM D I, ADAMS J H, JENNETT B (1975) Patients with head injury who talk and die. *Lancet*, **ii,** 375-377.

ROSE J, VALTONEN S, JENNETT B (1977) Avoidable factors contributing to death after head injury. *British Medical Journal*, **2,** 615-618.

SHINOHARA M, DOLLINGER B, BROWN G, RAPOPORT S, SOKOLOFF L (1979) Cerebral glucose utilization: local changes during and after recovery from spreading cortical depression. *Science*, **203,** 188-190.

SMALL J M, WOOLF A L (1957) Fatal damage to the brain by epileptic convulsions after a trivial injury to the head. *Journal of Neurology, Neurosurgery and Psychiatry*, **20,** 293-301.

SNOEK J, JENNETT B, ADAMS J H, GRAHAM D I, DOYLE D (1979) Computerised tomography after recent severe head injury in patients without acute intracranial haematoma. *Journal of Neurology, Neurosurgery and Psychiatry*, **42,** 215-225.

SPELLER R D, WHITE D R, SHOWALTER C K, ROTHENBERG L N, PENTLOW K S, MORGAN T J, SHOPE T B (1981) An evaluation of CT systems from ten manufacturers. *British Journal of Radiology*, **54,** 1053-1061.

TEASDALE G, JENNETT B (1974) Assessment of coma and impaired consciousness. A practical scale. *Lancet*, **ii,** 81-84.

TODOROW S, FELLER A-M (1982) Ueber das Vorkommen benigner sekundärer Bewusstseinstrübung—das Einschlafsyndrom—nach Schädel-Hirn-Trauma bei Kindern. *Zeitschrift für Kinderchirurgie*, **36**, 83-87.

WAGA S, TOCHIO H, SAKAKURA M (1979) Traumatic cerebral swelling developing within 30 minutes after injury. *Surgical Neurology*, **11**, 191-193.

WALTON G L (1898) Subarachnoid serous exudation productive of pressure symptoms after head injuries. *American Journal of the Medical Sciences*, **116**, 267-275.

WALTON G L, BROOKS W A JR (1897) Observations on brain surgery suggested by a case of multiple cerebral hemorrhage. *Boston Medical and Surgical Journal*, **136**, 301-305

WEINSTEIN M A, DUCHESNEAU P M, MACINTYRE W J (1977) White and gray matter of the brain differentiated by computed tomography. *Radiology*, **122**, 699-702.

ZIMMERMAN R A, BILANIUK L T, BRUCE D, DOLINSKAS C, OBRIST W, KUHL D (1978) Computed tomography of pediatric head trauma: acute general cerebral swelling. *Radiology*, **126**, 403-408.

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Good

Diffuse cerebral swelling following head injuries in children: the syndrome of "malignant brain edema"

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✔ The commonest initial computerized tomography (CT) finding in head-injured children is bilateral diffuse cerebral swelling. Cerebral blood flow and CT density studies suggest that this swelling is due to cerebral hyperemia and increased blood volume, not to edema. The clinical history, course, and outcome of 63 children with this CT pattern are reviewed. Fourteen children had a Glasgow Coma Scale score of greater than 8; all made a complete recovery and follow-up CT scans were normal. Forty-nine children had Glasgow Coma Scale scores of 8 or less. Fifteen had a history of a lucid period following the initial unconsciousness. One of these children died of delayed brain swelling, the others recovered well with minimal neurological deficit. Thirty-four children were rendered immediately and continuously unconscious. There was a high incidence of second lesions on the CT scan, 50% of this group developed intracranial hypertension and five died. All of the others were in coma for periods ranging from weeks to months. Follow-up CT scans showed an extracerebral collection with a density of cerebrospinal fluid in 27% of the patients, and ventriculomegaly with large sulci in 35%, whereas this pattern was seen only once in those with a lucid period. The difference between those with and without a lucid period is related to the degree of primary diffuse impact injury to the white matter.

KEY WORDS • children • head injury • cerebral edema • computerized tomography • cerebral blood flow • hyperemia • cerebral blood volume

In pediatric patients, rapid neurological deterioration has been well described, beginning several minutes to several hours following head injury.[25,31] The clinical picture is similar to the syndrome observed with an expanding intracranial hematoma, yet a surgical mass lesion is rarely found. This secondary clinical deterioration, while frequently self-limiting,[25,31] may progress rapidly to coma and death.[30] As many as 50% of children who die following head trauma are conscious on admission,[13] and 75% of deaths occur in the first 48 hours.[32] With aggressive resuscitation in the face of secondary deterioration, the incidence of "talk and die" in the pediatric age group has been cut to zero in our experience[6,7] and in that of others.[4] This fact suggests that a reversible pathological sequence was responsible for these deaths. In children who are conscious, and who then rapidly become unconscious and die, pathological studies show diffuse generalized brain swelling with little evidence of brain injury.[1] Among all children who die from head injury, diffuse cerebral swelling manifested by obliteration of the intracranial cerebrospinal fluid (CSF) spaces and venous congestion is the commonest autopsy finding.[19] Diffuse brain swelling has been recognized for a long time to be a common cause of secondary deterioration, and the brain swelling has been attributed to brain edema often prefixed with the word "malignant" because of the inexorable clinical course of so many children with this syndrome. Adult patients who deteriorate following a lucid interval almost always have an intracranial clot as the cause of the secondary deterioration rather than diffuse cerebral swelling, whereas in children the reverse is true.

Another group of severely head-injured children more closely resemble adults with head injuries. Unconsciousness occurs immediately at the time of injury and, although the patient's clinical status may

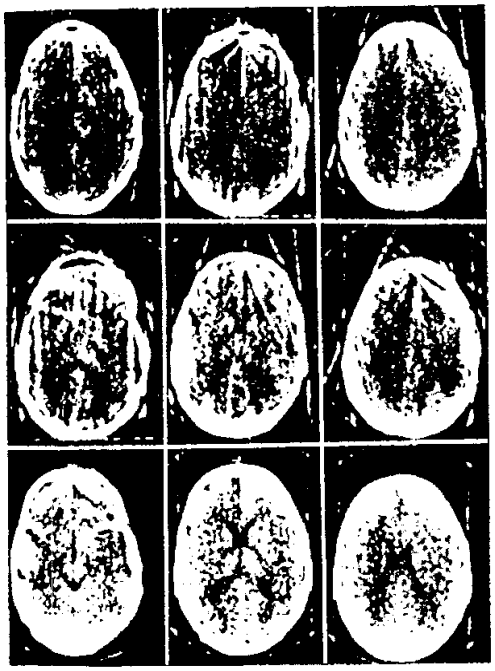

FIG. 1. Initial computerized tomography scans. *Upper:* Scan at 26 hours after injury shows diffuse swelling, small ventricles, and compression of the perimesencephalic cisterns. *Center:* Scan at Day 4 shows little change. The patient was still in coma. *Lower:* Scan at Day 26 shows return of the cisterns and ventricles to normal. The patient was awake and alert.

wax and wane, most of the injury to the brain appears to have been sustained at or shortly after the impact.[2,34] Presumably this group of patients have sustained a greater diffuse cerebral impact injury than the former group.

The purpose of this report is to document diffuse enlargement of the brain in children with the classical syndrome of "malignant" brain edema and present evidence that diffuse swelling in these circumstances is not, in fact, due to brain edema. Rather, it appears to be caused by severe cerebrovascular congestion. At this time, we are at a loss to explain the etiology of the vascular congestion based on knowledge of the control of the cerebral circulation in the normal state and under pathological conditions. The hyperemic syndrome is also documented in a number of children with no lucid period after trauma.

Clinical Material

Serial computerized tomography (CT) scans were performed on 214 children and adolescents, aged 6 months to 18 years, admitted to the Children's Hospital of Philadelphia (CHOP) between 1975 and 1979. Sixty-three of these patients had an initial CT scan performed in the first 24 hours after injury that showed evidence of diffuse cerebral swelling. The CT scan criteria for inclusion in the category of diffuse cerebral swelling are: 1) an initial CT scan showing small ventricles and cisterns with compression or absence of the perimesencephalic cisterns, and 2) a follow-up scan, 7 to 20 days later, showing return of the ventricular system and cisterns to normal size (Fig. 1).

The patients' clinical histories were reviewed for presence of a lucid interval (period of improved consciousness), early posttraumatic seizures, systemic shock (mean blood pressure below 50 torr), and evidence of other systemic injuries (such as long-bone fractures or ruptured viscus). The follow-up period was at least 2 months in all patients, and those with Glasgow Coma Scale (GCS) scores of 8 or less have been followed from the time of injury to present. All patients were graded using the GCS,[15] and 24 had a cumulative score of 5 or less, 25 had between 6 and 8, and 14 had greater than 8. Recovery was graded using the criteria of Jennett and Bond:[14] good recovery, moderate disability, severe disability, vegetative survival, and death.

The initial CT scan was reviewed for evidence of other cerebral lesions besides diffuse swelling. The follow-up scans were examined for the appearance of extracerebral collections of fluid; the appearance of abnormal, progressive ventricular enlargement, and evidence of enlarged sulci; and the resolution of this latter pattern with return to a normal CT scan. In 12 patients with serial, motion-free and artifact-free scans, all of which were performed on the EMI Mark I scanner,* the Hounsfield value of the deep frontal white matter on the initial scan was compared with that obtained after resolution of the diffuse swelling. The normal values of six children (aged 5 to 12 years) were calculated from a mean of 150 to 350 pixels taken from the deep frontal white matter.

The intracranial pressure (ICP) was monitored in 29 of the 63 patients, 21 with GCS scores of 5 or less, and eight with GCS scores of 6 to 8. A subarachnoid bolt was used in 28 cases and a ventricular cannula in one. All patients in whom ICP was monitored had an endotracheal tube in place and were receiving controlled hyperventilation to a $PaCO_2$ of approximately 25 to 30 torr at the time of insertion of the monitor.

The cerebral blood flow (CBF) was measured in six patients using the intravenous xenon-133 method[23] and a 16-probe detector system.† In all six patients, at least two CBF determinations were performed, one acutely and one after the diffuse swelling had resolved.[5] Three of these patients also had measurements of cerebral metabolic rate for oxygen ($CMRO_2$) concomitant with the measurements of CBF.

*EMI Mark I scanner manufactured by EMI Corp., Northbrook, Illinois.

†16-probe detector system manufactured by Harshaw Chemical Co., 6801 Cochran Road, Solon, Ohio.

TABLE 1

Incidence of subarachnoid hemorrhage (SAH)

Glasgow Coma Scale Score	Total Cases	Age (yrs)	SAH
3–5	24	5.4 ± 3.8	20
6–8	25	5.7 ± 4.4	11
>8	14	6.7 ± 4.5	1

Indium cisternograms were performed on five patients during the period when extracerebral collections were identified on CT scan. One child who developed hydrocephalus 1 year after trauma had a second indium cisternogram performed prior to any surgical therapy.[21]

Results

The CT pattern described as diffuse cerebral swelling was seen in 29% of all the children and adolescents studied. This pattern, however, was seen in only 15% of those with a GCS score of greater than 8, and in 41% of those with a score of 8 or less. Diffuse swelling occurred throughout the age range studied, which was 6 months to 18 years, and the mean age of patients with diffuse swelling was 5.9 years. There were 24 patients with a GCS score of 5 or less, 25 with a score of 6 to 8, and 14 with a score of greater than 8 (Table 1). The median GCS score was 7. The mean ages of the three groups of patients were not significantly different (Table 1).

Subarachnoid hemorrhage was the commonest associated finding, being demonstrated in 32 patients. Subarachnoid hemorrhage was seen as an area of increased density in the posterior portion of the interhemispheric fissure and occasionally over the tentorium. This finding was present in only 7% of the patients with GCS scores of greater than 8, in 44% of patients with scores of 6 to 8, and in 83% of patients with scores of 5 or less. Twenty-seven of the 63 (44%) initial CT scans showed evidence of another cerebral lesion (Table 2). These lesions ranged from depressed skull fractures through scattered small hemorrhages in the corpus callosum and deep white matter, which has been described as diffuse impact injury.[36] The type and frequency of the lesions are shown in Table 3.

In the 12 patients in whom CT Hounsfield values were measured, there was a significantly higher value on the initial CT scan showing diffuse swelling than on the follow-up scan ($p < 0.01$). Furthermore, the Hounsfield number of the deep white matter on the initial scan was higher than the normal range defined from the control hemispheres of children with normal CT scans (Table 4). This increase in Hounsfield density cannot be accounted for by an increase in water content of the tissues, as this would decrease the tissue density.[36] Measurements of regional cerebral blood volume demonstrated an increase in one patient with a return to normal values on recovery.[16] This same study did suggest that 48 hours or more after injury diffuse swelling could be seen in the absence of a documented increase in blood volume.

The six patients in whom CBF was measured showed an increased CBF (Table 5). The mean value for the fast component of CBF was not only higher than in patients with similar GCS scores and no diffuse swelling, but was also higher than in a control group of awake volunteers. Repeat CBF studies after resolution of the diffuse swelling in those patients who survived showed a decrease in CBF in all patients. Thus, a true hyperemia was present in association with the pattern of diffuse swelling. A measured decrease in CBF in response to a reduction in $PaCO_2$ was found in three patients in whom a CO_2 response was tested.

Intracranial pressure was measured in 21 patients with GCS scores of 5 or less and in eight with scores of 6 to 8. All patients were receiving controlled hyperventilation to a $PaCO_2$ between 25 and 30 torr at the time of insertion of the intracranial monitor. The initial ICP was > 20 torr in nine patients, and rose above 20 torr at some time in the first 3 days posttrauma in 17 of the 29 patients (59%). No patient died from uncontrollably elevated ICP, although 12 required maximum therapy: hyperventilation to a $PaCO_2$ in the low 20's (torr), osmotherapy to a serum osmolarity level of 320 mosmols or more, hypothermia to 32°C, and pentobarbital infusion to levels of 3 mg% or higher to maintain a normal ICP. In all patients the intensive care unit protocol was to maintain the ICP below 20

TABLE 2

Evidence of other lesions

Glasgow Coma Scale Score	Total Cases	CT Scan		Systemic Injuries		Shock		Seizures	
		No.	Percent	No.	Percent	No.	Percent	No.	Percent
3–5	24	13	54	10	42	4	17	0	0
6–8	25	8	32	6	24	2	8	6	24
>8	14	6	43	1	7	0	0	4	29

torr regardless of the arterial pressure and not to concentrate on cerebral perfusion pressure, since an adequate perfusion pressure in the damaged brain has never been defined.

Follow-up CT scans showed unilateral or bilateral extracerebral collections of CSF density in 17 patients (27%); none with GCS scores of less than 8, 24% with GCS scores of 6 to 8, and 46% with GCS scores of 3 to 5 (Table 6). These collections resolved without therapy in 16 of the 17 patients. In one patient, burr holes were made, and slightly xanthochromic CSF was obtained from both sides. The ICP was measured in eight of these patients during the period that the extracerebral collections were present, by lumbar tap in five, and by tap of the collections in three. In no patient was the ICP over 10 torr, and in those in whom the collection itself was tapped, the pressure in the collection was subatmospheric.

Cisternography was performed on five patients using indium ethylenediaminetetraacetate (EDTA). The collections were found to fill rapidly with the indium, although there was some mild delay in clearance of the isotope from the region of the sagittal sinus in three patients.[21] Three of the 17 patients with extracerebral collections developed progressive ventriculomegaly, two at 2 months, and one at 1 year after injury. These three patients had ventriculoperitoneal shunts inserted. The clinical condition of the children at the time of insertion of the shunt was vegetative or severely disabled in two who developed early ventriculomegaly. In these patients, the shunt had no effect on the clinical state. The third patient developed symptomatic hydrocephalus with vomiting and lethargy 1 year postinjury, at which time she had made a good neurological recovery. The insertion of the ventriculoperitoneal shunt led to complete resolution of her new symptomatology. These extracerebral collections seemed to not be a function of hydrocephalus nor to be subdural in site and, in general, required no surgical therapy.

Twenty-two (35%) patients developed a mild increase in ventricular size with enlarged Sylvian fissures and frequently also cortical sulci; none of these had GCS scores greater than 8, 32% had GCS scores of 6 to 8, and 58% had GCS scores of 3 to 5 (Table 6). In 10 of these patients, the ventricles returned to a

TABLE 3

Other lesions seen on computerized tomography

Lesions	No Lucid Period*	Lucid Period*	All Patients†
epidural	1	0	0
acute subdural	2	2	0
intracerebral hematoma or contusion	3	4	2
infarct	1	1	0
diffuse impact injury	5	0	0
linear skull fracture	0	0	3
depressed skull fracture	1	1	1

*Patients with Glasgow Coma Scale scores of 3 to 8.
†Patients with Glasgow Coma Scale scores of greater than 8.

TABLE 4

*White-matter density in patients with diffuse cerebral swelling and edema**

Group	No. of Cases	Hounsfield Value	Mean Change to Recovery
diffuse swelling	12	34.6–36.4	−3.2†
control	9	29.2–33.2	

*Computerized tomography findings.
†Significant at the $p < 0.01$ level (paired comparison t-test).

TABLE 5

Cerebral blood flow (CBF) and arterial blood gases in adults and children with acute head injury

Group	No. of Cases	$PaCO_2$	CBF-(F^1)* (ml/100 gm/min)
control	20	33	66
diffuse swelling (16–21 yrs)	6	35	75
no diffuse swelling (17–21 yrs)	8	35	43
adult	13	33	38

*F^1 = fast component of flow.

TABLE 6

Computerized tomography findings

Glasgow Coma Scale Score	Total Cases	Extracerebral Collection		Increased Ventricles		Decreased Ventricles	
		No.	Percent	No.	Percent	No.	Percent
3–5	24	11	46	14	58	7	29
6–8	25	6	24	8	32	3	12
>8	14	0	0	0	0	0	0

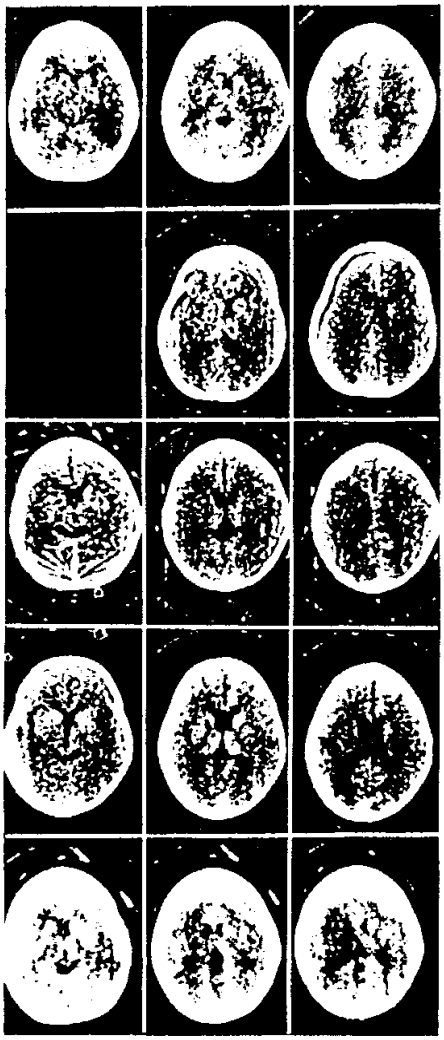

FIG. 2. Serial computerized tomography scans in a patient with a Glasgow Coma Scale score of 5 and no lucid interval. *Top Line:* Initial scan shows bilateral diffuse swelling. *Second Line:* Scan on Day 9 shows an extracerebral collection on the left. *Third Line:* Day 16. *Fourth Line:* Scan on Day 37 shows an atrophic pattern with large ventricles and Sylvian fissures. *Fifth Line:* Scan on Day 231 shows return of the brain to normal. Reproduced with permission from S. Karger AG, Basel, publishers, from Bruce DA, et al: Pathophysiology, treatment and outcome following severe head injury in children. *Child Brain 5:* 174–191, 1979.

normal size with a decrease in the size of the sulci within 6 months to 1 year after trauma (Fig. 2). Clinical recovery had usually begun by the time the ventricular enlargement was noted, and there was no close correlation between clinical recovery and return of the ventricles to a normal size. Subarachnoid hemorrhage was seen as frequently on the CT scans of the severely injured patients without diffuse swelling as it was on those with diffuse swelling, although no such pattern of extracerebral collections was seen in the former group. Ventriculomegaly was seen during recovery in 10 of 67 patients without initial diffuse swelling and cumulative GCS scores of less than 8. Diffuse enlargement was seen in only two, and this was minimal. Both of these children were shaken babies. Eight patients showed evidence of focal ventricular enlargement: five were shaken babies, and three had epidural hematomas. Intracranial hypertension was recorded in seven of the 10 patients. Thus, extracerebral collection and diffuse ventricular enlargement were not related simply to intracranial hypertension, subarachnoid hemorrhage, or GCS scores.

Early posttraumatic seizures (within the first 12 hours) were recorded in only 10 (16%) of the patients, and systemic shock (mean blood pressure less than 50 torr) in only six (9.5%). Other systemic injuries (such as abdominal bleeding or long-bone fractures) were present in 17 patients (27%) (Table 2).

Clinical History, Coma Score, and Outcome: Correlation With CT Findings

When the clinical history was reviewed, 23 patients (37% of the group) had a clear history of a lucid period following trauma. In eight patients with GCS scores of greater than 8, this lucid interval consisted of a period of talking and complete consciousness for minutes to hours after injury. There was then the onset of vomiting, headache, frequently pallor, and sweating associated with decreased alertness. The patients exhibited evidence of decreased spontaneous motor activity, loss of spontaneous speech, and loss of spontaneous eye opening but none progressed to become comatose (lack of eye opening and speech to deep painful stimulation). All of these patients made a good recovery, as demonstrated by normal follow-up CT scans.

Fifteen patients with an initial GCS score of less than 8 also had a well documented history of a lucid interval, defined as a period of improved consciousness. All of these children were unconscious following their trauma, then had a lucid period associated with recovery of eye opening, occasional words, and more spontaneous motor function. Rapid secondary deterioration occurred within minutes to hours after trauma, manifest by a deteriorating level of consciousness, loss of eye opening, and worsening motor responses, frequently with intermittent pupillary dilatation. Of this group of patients, 25% experienced episodes of apnea. A history of seizures was obtained in only 27% of the patients (Table 7), and systemic shock was not recorded in any of these patients. Eleven of these 15 patients had endotracheal intubation performed as part of their emergency resuscitation. One child in this group died as a result of delayed

TABLE 7

Relationship of lucid interval to shock, seizures, and other lesions

Consciousness*	No. of Cases	Age (yrs)	Shock	Seizures	Other Lesion on CT
lucid interval	15	6.3 ± 4.1	0	4	4
no lucid interval	34	5.2 ± 3.8	6	2	17

*All patients had a Glasgow Coma Scale score of less than 8.

TABLE 8

*Relationship of lucid interval to extracerebral collections, increased ventricular size, and outcome**

Consciousness	No. of Cases	Extracerebral Collections	Increased Ventricles	Decreased Ventricles	Outcome†				
					GR	MD	SD	V	D
lucid interval	15	1	1	1	14	0	0	0	0
no lucid interval	34	16	21	9	18	8	2	1	5

*All patients had suffered severe head injury and had a Glasgow Coma Scale score of less than 8.
†GR = good recovery, MD = moderate recovery, SD = severely disabled, V = vegetative, D = dead.

brain swelling. The others all made a good recovery. The mean hospital stay of this group was 14 days.

Thirty-four patients with GCS scores of 8 or less had no evidence of secondary deterioration and were immediately rendered unconscious. Thirty of these patients received endotracheal intubation as part of the emergency resuscitation. Seizures were recorded in 6%, and evidence of systemic shock was found in 18% (Table 7). Five patients in this group died, three were left in a severely disabled or vegetative state, and 26 made a good recovery or were moderately disabled (Table 8). The average hospital stay was 22 days, and 21 of the 29 survivors were transferred to a rehabilitation center following their acute hospital stay. The immediate unconsciousness, lack of a lucid period, longer hospital stay, and poorer recovery (Table 8) all suggest a greater degree of cerebral injury in these patients than in those with a lucid period, much of which might be accounted for by primary impact injury.[8] Indeed, four patients in this group had clear evidence of small scattered hemorrhagic areas within the deep white matter and corpus callosum on initial CT scan, and in three of the five patients who died, lesions of the corpus callosum or white matter were found at autopsy. These callosal lesions have been associated with diffuse impact injury to the brain.[2,34,36]

Table 6 demonstrates that the frequency of extracerebral collections and abnormally increased ventricular size and sulci is related to the initial GCS score. No child with a score above 8 demonstrated either abnormality. Table 8 shows the association of extracerebral collections and increased ventricular size in patients with a GCS score of 8 or less and the presence or absence of a lucid period. There is a highly significant correlation between the appearance of extracerebral collections, loss of cerebral substance as manifest by increasing ventricular and sulcal size, and the immediate onset of unconsciousness. Patients who developed secondary deterioration did not develop extracerebral collections or evidence of cerebral atrophy. There was no difference in the incidence of seizures or other systemic lesions between the two groups, although shock was more frequently encountered in the continuously unconscious patients.

Discussion

The use of serial CT scanning in a series of pediatric patients with severe head injury has allowed us to define an anatomical pattern that is seen in approximately 41% of such children. This pattern, which we have called "diffuse cerebral swelling," is rarely seen in the adult and, when it is seen, frequently involves only one hemisphere. The CT appearance is associated with a decrease in CSF and an increase in the bulk of the brain. There are only three components to the intracranial cavity: CSF, brain, and blood. Thus, an increase in brain bulk associated with a decrease in CSF spaces must be due to either an increase in brain water content or an increase in brain blood volume. A picture not dissimilar from that seen after head injury has been described in patients with pseudotumor cerebri. A decrease in ventricular volume is seen in these patients, and an increase in interstitial brain water content is believed to represent the major pathology and be the cause of the increase in brain bulk.[27,28] The CT scans in these patients, however, rarely show the lack of CSF in the cisterns, and

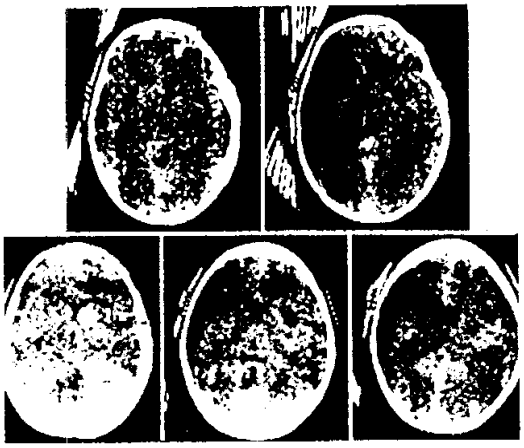

FIG. 3. Computerized tomography scans 3 days apart in a patient with a Glasgow Coma Scale score of 4 and no lucid period. *Upper:* Scan on Day 1 shows a depressed right frontal fracture with underlying hemorrhagic contusion, blood around the tentorium, and bilateral diffuse cerebral swelling. *Lower:* Repeat scan on Day 3 shows multiple areas of lucency scattered throughout both cerebral hemispheres consistent with cerebral edema. Some resolution of the diffuse swelling has occurred, as shown by the slightly larger ventricles. The area of decreased density under the bone of the left frontal area is an artifact since it is not seen on all cuts. Reproduced with permission from Bruce DA, et al: Acute brain swelling and cerebral edema in children, in de Vlieger, et al (eds): *Brain Edema.* New York: John Wiley and Sons, Inc, 1980 (In press).

never show the compression in the perimesencephalic cisterns seen in patients with brain swelling following trauma. Studies of regional cerebral blood volume in patients with pseudotumor cerebri demonstrate an increase in volume, but this was not considered adequate to account for the increase in ICP.[27] Measurements of CBF demonstrated a reduced CBF,[27] despite a normal metabolic rate and normal neurological function. While there is a superficial similarity between the CT picture of pseudotumor cerebri and that of diffuse swelling, a close comparison of the physiological studies reveals some major differences, which suggest a different underlying pathophysiology. In diffuse swelling after trauma, CT scan density is increased. A decrease in Hounsfield value that is linearly related to increases in water content of the brain has been demonstrated even under circumstances where increased protein content of the edema is found.[9,35] Thus, there is no evidence that brain edema is present on the initial scan in patients with acute, diffuse brain swelling. Cerebral blood flow studies have shown an increase in CBF, not the decrease seen in patients with pseudotumor cerebri. In one patient, studies showed increased cerebral blood volume in the acute phase.[16] Finally, the level of consciousness is invariably impaired, and the ICP may or may not be elevated, although we suspect intracranial compliance will always be reduced.

The increase in Hounsfield value, the clinical history of rapid deterioration, and the rapid recovery of the less severely injured patients all favor a transient, reversible physiological basis for the changes seen. Evans and Scheinker[11] and Langfitt, *et al.*,[18] have suggested that acute cerebral swelling is produced by vascular engorgement, and we conclude this appears to be the best explanation for the diffuse cerebral swelling seen following head trauma in children. Unfortunately, we do not have any insight into the basic triggering mechanisms of the diffuse swelling. Since this picture is seen across the spectrum of clinical injury, it cannot be directly related to the degree of primary brain injury. The mechanism of injury appears to be important since all the children had acceleration/deceleration type of injuries. This might imply that movement around the brain stem is the common factor involved in the production of the vascular engorgement. Several authors have demonstrated that brain-stem stimulation can increase CBF without increasing cerebral metabolism or altering the electroencephalogram.[17,20,26] Most recently, Raichle, *et al.*,[26] have suggested that locus ceruleus stimulation may change both CBF and cerebral capillary permeability, probably via central vascular aminergic pathways. Foltz, *et al.*,[12] have previously demonstrated that a concussive blow to the head may produce delayed conduction via the reticular formation, with evidence of a biphasic neuronal response. Thus, acute vascular changes may be precipitated by alterations in reticular formation or locus ceruleus function. These may be delayed in onset and may be independent of the degree of primary injury and dependent only on the mechanism of trauma.

Other explanations, however, may be considered. The most obvious is that of defective autoregulation. Although this possibility has not been specifically tested yet, we believe it is unlikely. A number of children who had minimal disturbance of consciousness and relatively minor injuries were seen and, when scanned, showed a pattern of diffuse cerebral swelling. It is difficult to believe that they had global loss of autoregulation. The ICP in unconscious patients with GCS scores of 5 to 8 was normal (under hyperventilation to 25 to 30 torr), despite a blood pressure that was above normal for the age. Indeed, in the select group of the most severely injured patients, a high blood pressure was associated with a high perfusion pressure (greater than 80 torr) and good outcome.[29] If autoregulation were defective, we would expect an elevated blood pressure to be associated with an increased ICP or a passive systemic arterial pressure (SAP)/ICP couple.

Other explanations, such as chemicals released by trauma into the CSF which percolate to reach the third or fourth ventricle and then trigger sudden vasodilation, cannot be ruled out. This has been shown to

occur with irritation within the third ventricle,[22] and can be produced by chemical stimulation of the brainstem areas discussed in the above section. Since we have no evidence for or against this explanation, it remains a possible one. Another explanation is that the hyperemia seen is a result of transient hypoperfusion and is a reactive hyperemia or is due to continuous seizures. Only a small percentage of our patients exhibit evidence of shock or seizure activity, and we consider this explanation unlikely. Finally, it is possible that the effects of trauma upon the cerebral vessels produce alterations in cerebrovascular tone which account for the hyperemia.[29] The reason why these vessels retain CO_2 responsiveness, and autoregulation is not lost, is that they are not truly flaccid vessels but simply have a new resting tone. There is evidence to show that systemic sympathetic stimulation can affect resting cerebrovascular tone and can affect response of the blood vessels to alterations in systemic arterial pressure.[3]

We conclude that the pattern of initial bilateral diffuse swelling following trauma in children is produced by vasodilatation and initial hyperemia. The increase in total intracranial blood volume may not be very high, since severe intracranial hypertension is rarely seen at the time of the initial CT scan. However, a redistribution of blood from the subarachnoid and pia vessels into the parenchyma would be adequate to account for the increase in brain bulk.

The clear difference in history, clinical presentation, acute and chronic course, and follow-up CT scans between patients with a GCS score of 6 to 8 versus those with a score of 3 to 5 requires an explanation. We believe that in the former group of patients, relatively little primary diffuse impact injury to the white matter has occurred. Therefore, when the hyperemia, decreased compliance, and high ICP are controlled, a rapid recovery occurs with little residual neurological damage, and follow-up CT scans show a rapid return to normal. In the second group, with a GCS score of 3 to 5 and immediate unconsciousness, a significant amount of primary impact injury to the white matter has occurred. The delayed rise in ICP at 2 to 3 days in this group is probably due to the presence of multifocal brain edema (Fig. 3) around areas of disrupted blood-brain barrier and small petechial hemorrhages. As the edema resolves, a loss of cerebral substance occurs with some evidence of CSF outflow obstruction leading to a period of increased CSF spaces. Extracerebral collections of CSF are seen that resolve spontaneously, leaving a pattern of apparent brain atrophy. In 43% of cases, presumably those with least axonal damage, reestablishment of brain bulk occurs. This event often occurs after physical recovery is well advanced but intellectual recovery is still underway. We have no proof of the mechanisms for this; possible explanations include a resolution of transient external hydrocephalus (unlikely because of the isotope scan findings), remyelination of white matter as occurs after experimental hydrocephalus,[19] and changes in brain protein composition or amount.[8] This concept of greater primary injury is supported by the time to recovery and limitation of recovery seen in the group of patients with GCS scores of 3 to 5, and by a recent pathological study on the brains of children who died after demonstrating diffuse swelling on CT scan.[33] By the time death occurred, areas of secondary ischemia were frequently seen, probably due to increased ICP.[33] The findings of diffuse impact injury are supported by the autopsy findings in our own patients.

We conclude that acute, diffuse brain swelling is very frequent in children and teenagers following acceleration/deceleration injury to the brain. The swelling is produced mainly by an increase in intracerebral blood, either as a true increase in cerebral blood volume or as a redistribution of intracranial blood from the pial to the intraparenchymal vessels. When associated with a lucid period, minimal underlying cerebral injury is likely, and hyperventilation for 24 to 48 hours may be all that is required. These children should all make a rapid and essentially complete recovery. In children in immediate deep coma, a significant degree of primary impact injury is present, and superimposed on this is the hyperemia. Thus, control of the swelling with hyperventilation is not adequate, and delayed increases in ICP occur due to true cerebral edema. These patients pass through a slow recovery with a series of specific CT appearances, but usually can be expected to recover to a self-sufficient state without further surgical intervention.

References

1. Adams H, Graham DI: Pathology of blunt head injuries, in Critchley M, O'Leary JL, Jennett B (eds): **Scientific Foundations of Neurology.** Philadelphia: F A Davis, 1972, pp 478–491
2. Adams H, Mitchell DE, Graham DI, et al: Diffuse brain damage of immediate impact type. Its relationship to "primary brain-stem damage" in head injury. **Brain 100**:489–502, 1977
3. Boisvert DPJ, Jones JV, Harper AM: Cerebral blood flow autoregulation to acutely increasing blood pressure during sympathetic stimulation, in Ingvar DH, Lassen NA (eds): **Cerebral Function, Metabolism, and Circulation.** Copenhagen: Munksgaard, 1977, pp 1.8–1.9
4. Bowers SA, Marshall LF: Outcome in 200 consecutive cases of severe head injury treated in San Diego County: a prospective analysis. **Neurosurgery 6**:237–242, 1980
5. Bruce DA, Obrist WA, Zimmerman RA, et al: The pathophysiology of acute severe brain swelling following pediatric head trauma, in Gotoh F, Nagai H, Tazaki Y (eds): **Cerebral Blood Flow and Metabolism.** Copenhagen: Munksgaard. **Acta Neurol Scand 60 (Suppl 72)**:372–373, 1979
6. Bruce DA, Raphaely RC, Goldbert AI, et al: Pathophysiology, treatment and outcome following severe head injury in children. **Childs Brain 5**:174–191, 1979

7. Bruce DA, Schut L, Bruno LA, et al: Outcome following severe head injuries in children. **J Neurosurg 48**:679–688, 1978
8. Corlen PL, Hofgate RL, Wortzman G, et al: Reversible cerebral atrophy in recent abstinent chronic alcoholics by computed tomography scans. **Science 200**: 1076–1078, 1978
9. Clasen RA, Huckman MS, Von Roenn KA, et al: Time course of cerebral swelling in stroke: a correlative autopsy and CT study, in Cervos-Navarro J, Ferszt R (eds): **Brain Edema. Advances in Neurology, Vol 28.** New York: Raven Press, 1980, pp 395–412
10. Epstein F, Rubin RC, Hochwald GM: Restoration of the cortical mantle in severe feline hydrocephalus: a new laboratory model. **Dev Med Child Neurol 16 (Suppl 32)**:49–53, 1974
11. Evans JP, Scheinker IM: Histologic studies of the brain following head trauma. I. Post-traumatic cerebral swelling and edema. **J Neurosurg 2**:306–314, 1945
12. Foltz EL, Jenkner FL, Ward AA Jr: Experimental cerebral concussion. **J Neurosurg 10**:342–352, 1953
13. Hendrick EB, Harwood-Nash DCF, Hudson AR: Head injuries in children: a survey of 4465 consecutive cases at the Hospital for Sick Children, Toronto, Canada. **Clin Neurosurg 11**:46–65, 1964
14. Jennett B, Bond M: Assessment of outcome after severe brain damage. A practical scale. **Lancet 1**:480–484, 1975
15. Jennett B, Teasdale G, Galbraith S, et al: Severe head injuries in three countries. **J Neurol Neurosurg Psychiatry 40**:291–298, 1977
16. Kuhl DE, Alavi A, Hoffman EJ, et al: Local cerebral blood volume in head-injured patients. Determination by emission computed tomography of ^{99m}Tc-labeled red cells. **J Neurosurg 52**:309–320, 1980
17. Langfitt TW, Kassell NF: Cerebral vasodilatation produced by brain stem stimulation: neurogenic control vs. autoregulation. **Am J Physiol 215**:90–97, 1978
18. Langfitt TW, Tannenbaum HM, Kassell NF: The etiology of acute brain swelling following experimental head injury. **J Neurosurg 24**:47–56, 1966
19. Lindenberg R, Fisher RS, Durlacher S, et al: The pathology of the brain in blunt head injuries of infants and children. **Proceedings of the Second International Congress of Neuropathology.** Amsterdam: Excerpta Medica, Part 2, 1955, pp 477–479
20. Meyer JS, Teraura T, Sakamato K, et al: Central neurogenic control of cerebral blood flow. **Neurology 21**:247–262, 1971
21. Moldofsky PJ, Alavi A, Zimmerman RA, et al: Radionuclide cisternography in cerebral trauma: correlation with computed tomography. (In press)
22. Nicolaïdis S, Hoa LV, Le Beau J: Étude hémodynamique isotopique de l'oedème cérébral expérimental aigu. Mécanisme physiopathologique. **Neurochirurgie 18**: 521–530, 1972
23. Obrist WD, Thompson HK Jr, Wang HS, et al: Regional cerebral blood flow estimated by ^{133}Xenon inhalation. **Stroke 6**:245–256, 1975
24. O'Keefe CM, Bruce DA, Downes JJ: ICP and cardiovascular function in pediatric head trauma. **Anesthesiology 51**:S316, 1979
25. Pickles W: Acute general edema of the brain in children with head injuries. **N Engl J Med 242**:607–611, 1950
26. Raichle ME, Eichling JO, Grubb RL Jr, et al: Central noradrenergic regulation of brain microcirculation, in Pappius HM, Feindel W (eds): **Dynamics of Cerebral Edema.** New York/Heidelberg/Berlin: Springer-Verlag, 1976, pp 11–17
27. Raichle ME, Grubb RL Jr, Phelps ME, et al: Cerebral hemodynamics and metabolism in pseudotumor cerebri. **Ann Neurol 4**:104–111, 1978
28. Sahs AL, Joynt RJ: Brain swelling of unknown cause. **Neurology 6**:791–803, 1956
29. Saunders ML, Miller JD, Stablein D, et al: The effects of graded experimental trauma on cerebral blood flow and responsiveness to CO_2. **J Neurosurg 51**:18–26, 1979
30. Scheinker IM: Vasoparalysis of the central nervous system, a characteristic vascular syndrome. Significance in the pathology of the central nervous system. **Arch Neurol Psychiatry 52**:43–56, 1944
31. Schnitker MT: A syndrome of cerebral concussion in children. **J Pediatr 35**:557–560, 1949
32. Sevitt S: Fatal road accidents in Birmingham: times to death and their causes. **Injury 4**:281–293, 1973
33. Snoek J, Jennett B, Adams JH, et al: Computerized tomography after recent severe head injury in patients without acute intracranial haematoma. **J Neurol Neurosurg Psychiatry 42**:215–225, 1979
34. Strich SJ: Shearing of nerve fibres as a cause of brain damage due to head injury. A pathological study of twenty cases. **Lancet 2**:443–448, 1961
35. Torack RM, Alcala H, Gado M, et al: Correlative assay of computerised cranial tomography (CCT), water content and specific gravity in normal and pathological postmortem brain. **J Neuropathol Exp Neurol 35**: 385–392, 1976
36. Zimmerman RA, Bilaniuk LT, Gennerelli T: Computed tomography of shearing injuries of the cerebral white matter. **Radiology 127**:393–396, 1978

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Figure 6.68 Base of the brain illustrating a dense basal subarachnoid hemorrhage that centers on the circle of Willis. This victim was involved in a bar fight in which he was hit with a single violent blow to the side of the face and fell backward, striking his head. He was dead upon arrival to the hospital. A cursory examination did not reveal an obvious aneurysm, though it was thought a posterior communicating artery was torn. This case illustrates a typical example of traumatic vascular injury.

posterior impacts. The context for such injuries is most commonly forceful rotation of the head on the neck.

Traumatic Cerebral Edema

Whenever there has been physical cerebral or spinal cord injury, some degree of edema will inevitably occur in the vicinity of the lesion. If one subscribes to the classification of vasogenic and cytotoxic forms of edema (discussed in greater detail in Chapter 5), some disruption of the blood-brain barrier quite early will result in vasogenic or extracellular edema. If this edema is not compensable, its mass and pressure effects may affect vascular perfusion in the region, resulting in secondary ischemia of the region, which will alter metabolism in nearby cells, resulting in cytotoxic or intracellular edema. This form of edema may also be produced by release of neurotoxic products of inflammation or cell injury that alter cell membrane function and may irreversibly damage affected cells, usually neurons. Regardless of which form of edema exists in response to trauma, it may spread well outside the traumatized region and involve the whole hemisphere or whole brain. In this instance, there is a grave risk of excessive and irreversible increases in intracranial pressure, herniation, and perfusion failure, which may lead to brain death.

Traumatically induced edema, from whatever cause, is always capable of expanding the intrinsic mass effects of any lesion present in the adult, but in the child who has suffered head trauma, cerebral edema may occur in connection with even apparently mild injury and may lead to death [282, 283]. A typical example of this phenomenon may be seen in

the child who falls from a window to the pavement or is struck by a vehicle, who may or may not suffer a skull fracture and may or may not suffer loss of consciousness, but within hours of the traumatic episode may become stuporous and drift into coma from elevated intracranial pressure. This phenomenon has been observed for many years by emergency room physicians and neurosurgeons but has not been satisfactorily explained. A possible explanation may lie in the fact that cerebral blood flow in response to injury differs with age. In children under the age of 5 years, impacts to the head may result in increased cerebral blood flow, whereas in the adult the response may be just the opposite [284–287]. With increased blood flow into a possibly damaged vascular bed, the blood-brain barrier may be more likely to open, giving rise to massive edema, which may or may not be fatal. The importance to the pathologist of this phenomenon is when attempting to develop a mechanism for death with the confusing and seemingly inconsistent finding of little evidence of brain trauma in the face of massive edema, with no obvious anatomic cause. Suffice it to say that even though the explanations are not satisfactory, this phenomenon has been observed on a regular basis in forensic pathology practice, and one should not hesitate to assign a traumatic cause to such deaths, even though a lucid interval may have occurred.

In the adult, cerebral edema of a traumatic origin, if long standing, may have deleterious effects on myelin that may lead to demyelination [241]. Altered metabolic states such as prolonged acidosis, or associated conditions such as fat embolism, and the basic nature of shearing forces that might have injured long axons of passage can also be satisfactory explanations for apparent demyelination about contusions or deeper brain lesions, but some workers feel that at least some of the demyelination observed (discussed above) in some cases is due to edema alone. This contention is difficult to prove or disprove.

Pulmonary Edema in Connection with Head Trauma

In some cases of head trauma, usually severe, the victim may suddenly appear to suffer cardiac failure as evidenced by severe massive pulmonary edema [288]. The basis for this edema appears to have at least some neurogenic component but may interact with other factors, such as disseminated intravascular coagulation, fat embolism, hypovolemic shock, electrolyte disturbances, and thoracic injuries that may also affect myocardial function [289–291]. The mechanisms of so-called neurogenic pulmonary edema are not yet understood, but trauma may not be the only underlying condition giving rise to the phenomenon. An association with epilepsy and the sudden unexpected death in connection with seizures has also been reported and is discussed in Chapter 9 [292].

Post-Traumatic Demyelination

After any destructive lesion of the cerebral cortex, it is inevitable that there will be some reflection of loss of axonal input to the white matter by pallor of myelin beneath the lesion. This myelin pallor is usually lost as sections are examined at greater and greater distance from the lesion. Often this pattern of pallor is greatest about the ventricles, as described by Grcevič and many others [203], and in other instances the loss is apparently quite diffuse, involving much of the centrum ovale or temporal lobe white matter (see Figure 6.65), and is quite independent of the degree of cortical pathology [158, 241, 258]. In such cases, the degree of demyelination is so evident that the appearance closely resembles myelin loss as seen in one of the leukodystrophies, with sparing of the subcortical U fibers. The basis for this pathological appearance is probably locally extensive axonal injury from shear or stretching forces (traumatic epicenter) as well as by myelin loss caused by secondary

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TAB 13

ACE COURT REPORTING SERVICE

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Statewide & Nationwide Scheduling

ORAL DEPOSITION OF

Norma J Farley M.D.

Taken on November 20, 2007

Cause No. 07-CR-885-B; State of Texas v. Melissa Lucio; in the 138th District Court, Cameron County, Texas

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CAUSE NO. 07-CR-885-B

STATE OF TEXAS	§	IN THE DISTRICT COURT
	§	
Plaintiff	§	
	§	
VS.	§	138TH JUDICIAL DISTRICT
	§	
MELISSA LUCIO	§	
	§	
Defendant	§	CAMERON COUNTY, TEXAS

ORAL AND VIDEOTAPE DEPOSITION OF

NORMA JEAN FARLEY, M.D.

ORAL AND VIDEOTAPE DEPOSITION of NORMA JEAN FARLEY, M.D., produced as a witness at the instance of the State and duly sworn, was taken in the above-styled and numbered cause on November 20, 2007, from 3:03 p.m. to 3:27 p.m., before Annette E. Escobar, Certified Shorthand Reporter in and for the State of Texas, reported by computerized stenotype machine at the offices of 197th District Court, 977 E. Harrison, Cameron County Courthouse, Brownsville, Texas, pursuant to the Texas Rules of Civil Procedure and the provisions stated on the record or attached hereto.

APPEARANCES:

ATTORNEYS FOR THE STATE:

Hon. Joe Kripple
Hon. Mary Jane Zamarripa
Hon. Alfredo Padilla
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-- and

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(956) 399.1299

ALSO PRESENT:

Joe Lopez, Cameron County Investigator, Videographer

INDEX

EXHIBITS

P-R-O-C-E-E-D-I-N-G-S

Whereupon,

3:02 PM

MR. KRIPPLE: Joe Kripple on behalf of the State, present.

MS. ZAMARRIPA: Mary Jane Zamarripa on behalf of the State, present.

MR. PADILLA: Alfredo Padilla on behalf of the State, present.

MR. GILMAN: Peter Gilman on behalf of the Defendant.

MR. CORDOVA: Adolfo Cordova on behalf of the Defendant.

MR. GILMAN: And for the record the Defendant is present.

NORMA JEAN FARLEY, M.D.,

after having been first duly sworn, was examined and testified as follows:

EXAMINATION

BY MR. GILMAN:

Q. Dr. Farley, I've asked for this deposition. Could you please state your name for the court record?

A. Yes, it's Dr. Norma Jean Farley.

Q. And you performed the autopsy in this particular case; is that correct?

A. Yes, I did.
Q. And could you give us the date that you performed this autopsy?
A. Yes, 2-19-2007 was the day that we opened the body, 8:05.
Q. And when did you receive this body?
A. I know it was there Sunday somewhere between Saturday and Sunday the decedent died. The external examination of looking at the body and all of that is part of the autopsy that was mainly done on the 19th.
Q. And the body that you had, you received is whom?
A. Maria Alvarez.
Q. And she is?
A. Two year old female.
Q. Doctor, what I am hoping that we can go through today is your findings, your conclusions that you made as a result of performing this autopsy, and I was thinking about starting at the ahead and going to the arms, the torso and then the legs and then the internal parts if we don't cover them in between. Okay?
A. Yes. It's in the autopsy report pretty vividly and there's a lot of contusions, so this is going to take a long time.
Q. I think it's important that we find out the full extent of these injuries, doctor, okay.

Can we start with the head of this two year old child and explain to the jury and what your findings were.

A. Well, if you look at the final principal diagnosis that's the final findings. And basically, it's blunt force head trauma. There's evidence of blunt force head trauma. There's subarachnoid hemorrhage, there's subdural hemorrhage. Basically subarachnoid hemorrhage means blood directly around the brain. Subdural hemorrhage means blood between the brain and skull so there is blood where there shouldn't be in the skull. There was also swelling in the brain, cerebral edema. Basically the brain is swollen and it shouldn't be. It should be normal and there's swelling. We found multiple scalp contusions, and, of course, there are multiple contusions and abrasions all over the body.

Q. Okay. Can you explain to the jury what these contusions and abrasions what does that mean?

A. A contusion is a bruise, basically. It's due to trauma. Usually most people can relate if someone falls down and gets hit and then they get this discoloration of the skin due to hemorrhage into the soft tissue or the skin itself it will cause a bruise or a contusion.

Abrasion is what common people might call a scrape.

Q. Okay. Could you make a determination of the cause of these abrasions, contusions and the blood excess of blood around the brain?

A. Yes, that's what blunt force head trauma means, means beat about the head or thrown or hit up against an object, basically; beaten.

Q. And so that we can, a jury can understand this how would, how would this come about?

A. Beating. I don't know how else to make it clear. Beating.

Q. With a hand, a fist, a club?

A. Whatever. We don't know for sure what was used to hit the child, but for sure there's bruises and contusions. We don't see things that we would call a pattern abrasion on the head like from a broom where you get parallel lines or something like that.

So all we know that they were beaten. They could be a hand, yes, could be a fist or you can actually take a child and beat their head in with something and that's also possible.

Q. Okay. Anything else about the head?

A. No. And if you go under external examination, there's a whole paragraph describing all these contusions and abrasions to the head which the jury will be able to read for themselves.

Q. Okay. Let's go to the arms.

A. Now, do you want me to go from the findings, from the -- or the whole paragraphs on the arms.

Again --

Q. The more -- the more -- the more you can tell us about the extent of these injuries, the better it's going to be for my client and for us. We are not doctors and we need to know the full extent of those injuries.

A. Well, it's very in depth in the autopsy report. There's a page of, on the extremities describing all the different contusions I saw on the body that day. Also describing different abrasions that were also on the body. There are also pattern blank abrasions, kind of parallel looking abrasions, suspicious for, like, a slap or finger marks. There was also a left humerus fracture which means the arm was broken on the left. It's a spiral type fracture which was also seen on the x-rays. A bunch of crusted abrasions.

I mean, there's a lot. Someone can read it from my autopsy if you want to, but basically just telling you the different types of bruises, colors of bruises, but they're everywhere.

Q. Okay. A broken bone, explain to the jury this broken bone how it occurred, if you know?

A. (Nods head). I don't know how it occurred, if

it was twisted. There is usually a torsion-type injury where you grab someone and kind of twist the arm. It's called a spiral fracture. We often see it in abused cases in children from someone grabbing and twisting violently the arm.

It goes along with all the other findings or finding on this child of all these contusions and abrasions, and, of course, there's same kind of marks on this arm, so there's actually a fracture there. Children are usually very resilient and don't fracture. And this is a particular type spiral fracture that's highly suggestive and suspicious for abuse.

Q. How old a fracture is this?

A. I can't say for sure it is a healing fracture. Children heal with different stages than adults do. In fact, most people know that their kids' bruises and injuries seem to heal faster than an older person.

It's a healing one. It's sort of got fibrosis and granulation in the wound. It's probably several days.

Q. So we're talking days, we're not talking months?

A. No, not months.

Q. Okay. More than a week?

A. You can't get that precise with these kind of fractures in children.

Q. Okay. Were there any -- in your findings were there anything that related to more than a week old?

A. Most of the injuries were probably within the last week. I mean, definitely the head trauma. When we say acute, we mean it was fairly quickly in the last 24 hours most likely for the head trauma. And the acute hemorrhage we're seeing in the skull itself.

The other injuries like contusions and stuff, they can go out a week or so and be green, green-yellow brown. Again, you take microscopic sections and you try to see if there's any pigment or called hemosiderin to see if they're older; in this case there were some older wounds. We could tell that by looking too but nothing that's going out three and four weeks. Children tend to heal faster than that.

Q. The bruising, the contusions, this broken bone, broken bone didn't cause the death?

A. No.

Q. The cause of the death was the injury to the head?

A. Yes.

Q. And the injury to the head happened within how much time, do you think?

A. It's hard to say. It's acute. The blood that we're seeing in the subdural space doesn't appear to have

any fibroblast or pigment I'm talking about. So I say it was acute. The neuropathologist also said acute, which means usually, you know, within a day or so they start to organize after that. But the exact time we don't know. The child would have had symptoms, in my opinion, immediately after staining this type of trauma. But the exact time frame in that 24-hour period I can't give you.

Q. Okay. And just so that we understand and the jury can understand, when you say within 24 hours, 24 hours of the death?

A. Yes.

Q. Not twenty-four hours from the time that you --

A. Death. The child doesn't keep bleeding after it dies. The heart doesn't pump anymore.

Q. All right. The torso, explain to this jury what you found in the torso?

A. The same. There are numerous contusions and abrasions. There are also bite marks up on the right back. These bite marks have contusions, but they're also, like, dragging. Looking like dragging of the teeth. There's abrasions associated with them, and if I believe they're on the right back. But again multiple, multiple contusions on the trunk. The trunk would be the abdomen, chest and back. Lots of contusions again.

Q. The bite marks these are human bite marks?

A. In my opinion, they are. There's bite marks on the right upper back. Unfortunately, for an odontologist to tell you and match somebody with them, you have to actually have to leave an impression for them to be able to match, too. In this case they're dragged, they're abraded and there was no way to try to match them.

Q. Okay. How much force is required to leave such marks on a child two years of age?

A. This -- this -- these were pretty bad. I mean, they're abrasions with them. If you picture a child biting someone they leave little impressions of teeth. This actually left blood in the soft tissue as well as a scrape on the surface. So it's not like a little kid just kind of biting and leaving a tiny little teeth impression. This left an abrasion like you get if your knee went across the asphalt.

Q. In the torso, is there any internal injuries?

A. Yes, there were bruises or contusions to the lungs and there was also contusion to the right kidney.

Q. Explain how I would get bruises to my lung and kidney?

A. Again, that would be consistent with being beat with an object or objects or kicked or stomped or, you know, however you get the beating that's how it would occur.

Q. And the time frame from the death?
A. I can't tell you that. I don't know for sure.
Q. Estimated?
A. Unh-unh.
Q. Okay.
A. It didn't kill the child. It's just another sign of the amount of injury to the child.
Q. The injuries that this child sustained, did it come all of a sudden at one time, the beating, or is this maybe beatings that took place over a period of time?
A. In my opinion, the child did have signs that it had been beaten over a period of time. The cause of death is a blunt force head trauma, and this time whoever beat the child beat it enough to kill it when they hit it in the head.
Q. And death was it instant or lingers or what?
A. I wouldn't know. You can go in a coma. What I'm saying is when it got this kind of injury there would have been symptoms immediately, signs and symptoms of the trauma. Either they're lethargic like they're trying to fall asleep or they go into a coma, but they wouldn't be acting normally after this type of head injury. Typically, that's what happens, they'll seize a little bit. They'll be lethargic or sleepy then they might seize a little bit. Every kid is a little bit different. They

usually slip into a coma or die.

Q. The injuries to the legs, ma'am.

A. That's the same thing as extremities. The same kind of -- when I explained that there were abrasions and pattern abrasions I was explaining the legs, too. The extremities would be the upper and the lower extremities.

Q. You have a diagram on your autopsy report that shows that there's, looks like some parallel marks on the inner thigh of the left leg.

A. Yes. That's what I was talking about when I said there were these parallel contusions that were patterned that looked like a slap or a hit. That's what I was talking about when you asked me about extremities earlier.

Q. Okay. There's also on the diagram it looks like you've noted a contusion on the right bottom of the foot.

A. Yes.

Q. Can you --

A. And a laceration which is a tear.

Q. And how is -- I mean is that a beating on a foot or is that walking on --

A. I wouldn't know. I just put what I found.

Q. Sure.

A. Given all the other injuries I can't rule out that it's abuse, but she stepped on thorn or something I

can't rule that out.

Q. Okay. On the next page you have a picture of the vaginal area.

A. Sorry, I don't have the same report you do, so they're not in the same. I have the same report but not in the same order. Go ahead and talk about it. I'll remember it.

Q. Well, you've indicated abrasions and a couple of contusions. Could you describe your findings there?

A. Yeah. I think they're just nonspecific. I didn't see any injury to the hymen in the opening of the vaginal oraface or the vagina itself. So I didn't see obvious evidence of sexual abuse, but I went ahead and marked what I found.

Q. Okay. Doctor, in your -- in your job in dealing with a child like this, are there tests made, like are the bones extremely brittle? Is this a type of person who bruises easily? Are there any kind of tests that are made?

A. Not postmortem, no, not usually. The blood hemolyzes, so we can't do certain tests for coagulation disorders. Generally x-rays point out most of the brittle bones or other bone abnormalities because you see all the different places where the bone is slightly fractured and it healed itself.

Also, people with brittle bones they tend to have blue sclera, blue, blue, and this child did not have that at all. And you would expect more fractures in different places with brittle bones. This is one fracture in one place. Now, I'm not even -- it wouldn't be brittle bones in this child.

Q. Okay. Were there any other fractures of the bones?

A. No.

Q. Just this one bone?

A. Yes, just this one.

Q. Doctor, could you go in more for a moment, the symptoms that if this child received some blunt trauma that caused eventually the death, what kind of symptoms would be noticeable that people should be able to pick up on?

A. That's what I had mentioned earlier, the lethargy, the tiredness, the unable to wake them up. They twitch and seize. Sometimes some of our children with blunt head trauma they're actually diagnosed at the ER as seizure disorder and it has nothing to do with the, their seizing because there's blood around the brain.

They wouldn't be able to wake them up. They wouldn't be acting like they should and they'd be breathing funny. Usually, people notice gurgling,

abnormal breathing and you can't wake them up if they go into a coma.

Q. Okay. Were you able to determine, roughly, the time of death?

A. Well, the child I think they pronounced actually at the hospital.

Q. Okay. But she had actually died at home?

A. It doesn't matter. They pronounce, wherever they pronounce is when they die.

Q. Okay. And you're saying that within 24 hours from the time they pronounced it is roughly the time within that time frame is the head trauma?

A. Yeah, that's just an estimate.

Q. Sure.

A. Because they can lay there in a coma.

Q. Sure.

A. But by looking at the smears, the subdural hemorrhage, it appears to be an acute response, meaning that it didn't happen two or three days ago. It's something acute.

MR. GILMAN: That's it.

MR. KRIPPLE: You want to take five minutes just to collect your thoughts and make sure you've asked her everything?

MR. GILMAN: If you don't mind.

MR. KRIPPLE: Easier to take a couple minutes now than to do this again. We'll go off the record.

MR. GILMAN: Sure.

(Discussion off the record was had.)

Q (By Mr. Gilman) Doctor, you took some pictures during your autopsy, did you not?

A. Yes, I did.

Q. And did you take a picture of the overall body of this little girl?

A. Yes, I did.

Q. And do you have that in your...

A. Yes, and I think you have a bigger one, don't you?

MR. KRIPPLE: We may have any of the pictures you're talking about.

A. I have one here.

MR. KRIPPLE: Refer to it and we can put it into evidence.

A. I have so many of these. Let me get -- this is pretty decent, too. It's not overall. But this is linear marks you were asking me about. They're all stuck together. This is pretty good, too. The bite marks you asked about, same one. See what I mean? There we go.

MR. GILMAN: If I can mark those.

A. You want one of these.

MR. GILMAN: Yeah.

MR. KRIPPLE: No objection. They came out of her file.

Q (By Mr. Gilman) Doctor, these are pictures that you've given me that you had taken during the autopsy and they're marked 1 through 9.

Do these fairly and accurately depict the injuries that this child sustained during this --

A. Yes, and these are the external injuries. There's internal ones, too, but that depicts the external.

MR. GILMAN: I'd like to put these into evidence.

MR. KRIPPLE: Sure.

MR. GILMAN: That's all I have. Thank you. Thank you, doc.

THE WITNESS: Sure.

MR. KRIPPLE: State reserves all questions.

(Signature having been not waived, the deposition was concluded at 3:27 PM.)

ERRATA SHEET -- CHANGES AND SIGNATURE

PAGE	LINE	CHANGE	REASON

NORMA JEAN FARLEY, M.D.

WITNESS SIGNATURE

I, NORMA JEAN FARLEY, M.D. have read the foregoing deposition and hereby affix my signature that same is true and correct, except as noted above.

_____
NORMA JEAN FARLEY, M.D.

THE STATE OF TEXAS §
COUNTY OF CAMERON §

Before me, _____, on this day personally appeared NORMA JEAN FARLEY, M.D. known to me or proved to me on the oath of _____ or through _____ (description of identity card or other document) to be the person whose name is subscribed to the foregoing instrument and acknowledged to me that he/she executed the same for the purpose and consideration therein expressed.

Given under my hand and seal of office on this _____ day of _____, 2007.

_____
NOTARY PUBLIC IN AND FOR
THE STATE OF TEXAS

My Commission Expires: _____

CAUSE NO. 07-CR-885-B

STATE OF TEXAS	§	IN THE DISTRICT COURT
Plaintiff	§	
VS.	§	138TH JUDICIAL DISTRICT
MELISSA LUCIO	§	
Defendant	§	CAMERON COUNTY, TEXAS

REPORTERS CERTIFICATION
OF NORMA JEAN FARLEY, M.D.
NOVEMBER 20, 2007

I, Annette E. Escobar, Certified Shorthand Reporter in and for the State of Texas, hereby certify to the following:

That the witness, NORMA JEAN FARLEY, M.D. was duly sworn and that the transcript of the deposition is a true record of the testimony given by the witness.

That the deposition transcript was duly submitted on December 6th, 2007, to the witness or to the attorney for the witness for examination, signature, and return to me by December 21st, 2007.

That pursuant to information given to the deposition officer at the time said testimony was taken, the following includes all parties of record and the amount of time used by each party at the time of the deposition:

PETER GILMAN (00:25 MINUTES), Attorney for Defendant.

That a copy of this certificate was served on all parties shown herein on _____ and filed with the Clerk.

I further certify that I am neither counsel for, related to, nor employed by any of the parties in the action in which this proceeding was taken, and further that I am not financially or otherwise interested in the outcome of this action.

Further certification requirements pursuant to Rule 203 of the Texas Code of Civil Procedure will be complied with after they have occurred.

Certified to by me on this 26th day of November, 2007.

ANNETTE E. ESCOBAR, CSR-RPR
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Hilda Y. Thompson,M.D.
Wm.Eddy,HT,CT(ASCP)CT(IAC)

AUTOPSY REPORT

Mail to:

Judge Sallie Gonzalez
608 East Harrison
Harlingen, Texas 78550

Patient Name:	**ALVAREZ, MYRIAH**		
MRN:	MORGUE-5526	Case #:	OA07-38
DOB/Age:	9/6/2004 (Age: 2)	Account #:	VBMC MORGUE
Gender:	F		
Location:	VBMC MORGUE	Autopsy Date:	2/18/2007 08:05
Ordered By:	Judge Sallie Gonzalez	Reported:	9/19/2007
Copy To:	Armando Villalobos		

Authorized By: Justice of Peace Sallie Gonzalez

Performed By: Norma Jean Farley, M.D.

Final Principal Diagnoses:

I. Blunt force head trauma.
- A. Subarachnoid hemorrhage, acute and multifocal.
- B. Acute subdural hemorrhage (approximately 25 cc) with intradural hemorrhage.
- C. Anoxic-ischemic encephalopathy, diffuse.
- D. Cerebral edema.
- E. Multiple scalp hemorrhages.

II. Eye pathology (please see Eye Pathology Report).
- A. Traumatic iris pigmentation of anterior lens capsule.
- B. Bilateral fixed retinal folds.
- C. Bilateral disruption internal limiting membrane.
- D. Bilateral perineural hemorrhage.
- E. Findings highly suggestive of non-accidental trauma.

III. Evidence of battered child syndrome.
- A. Bite marks on right upper back (2).
- B. Numerous contusions and scattered abrasions involving the head, trunk and extremities.
  1. Patterned abrasions on right calf and left thigh.
- C. Subdural hemorrhage cervical and thoracic spinal canal.
- D. Right foot laceration and contusion.
- E. Left humerus fracture, healing.
- F. Bilateral lung contusions.
- G. Right kidney contusion.
- H. Subcutaneous hemorrhage, right buttock.
- I. Right great toe nailbed hemorrhage.
- J. Multiple crusted abrasions involving the scalp with thinning of the scalp hair.
- K. Remote frenulum injury.

IV. Normally developed, thin, female child.

A. Dehydration with poor skin turgor, sunken eyes and confirmed by vitreous electrolytes.
B. Child at < 5th percentile by body height and approximately at the 30th percentile by weight.
C. Pulmonary edema and congestion.

Conclusion: It is my opinion that Myriah Alvarez, a 2-year-old female child, died as the result of blunt force head trauma. At autopsy, there is evidence of significant blunt force head trauma with multiple contusions and abrasions involving the head, multiple scalp hemorrhages, cerebral edema, and subarachnoid and subdural hemorrhage (blood around the brain and blood between the brain and skull). The findings in the eyes are also highly suggestive of non-accidental trauma. Other autopsy findings are listed above and include contusions involving both lungs and the right kidney, a healing fracture of the left humerus and multiple abrasions and contusions to the body. Dehydration is present and confirmed by vitreous electrolytes. The manner of death is homicide.

Electronically Signed Out
Norma Jean Farley, M.D.

Gross Description:

General Examination

The body is that of a normally developed, thin, female child, 82.8 cm tall and weighing approximately 27 pounds. The decedent is nude at the time of initial examination.

External Examination

Rigor mortis is fully developed in the extremities, jaw and neck. Unfixed livor mortis is found on the dorsal dependent portions of the body. The decedent has dark brown scalp hair that has blotchy areas of thinning with multiple crusted abrasions present, most prominent in the right parietal and occipital scalp. There are multiple contusions present on the body that will be described under evidence of injury. The eyes are open and sunken; the conjunctivae are pale and without petechiae; the corneae are clear and the irides are brown. Skin turgor is poor. The ears, nose and external auditory canals are normally developed. The teeth are natural and decidual teeth are present.

The neck is straight and free of indentations. The thorax is symmetrical and the abdomen is retracted. The back is straight. On the lower left back is a V-shaped 0.8 cm scar. The anal opening is unremarkable. On the left buttock is a linear 1.3 cm white scar. On the right back is a 3 mm curvilinear white scar. The genitalia are those of a normal female child. The hymen is slightly open and a previous sexual assault kit has been taken prior to the autopsy.

The upper and lower extremities are symmetrical and normally developed. There are two off-white scars noted on the ventral left forearm measuring 1 and 0.4 cm. On the distal dorsal left forearm are scattered small white scars measuring up to 0.6 cm. On the dorsal right arm are three white scars measuring from 0.1 to 0.9 cm. A 2 mm scar is noted on the ventromedial distal right forearm. Linear and V-shaped scars are noted on the left hand measuring up to 1.8 cm. The fingernails are short to intermediate in length with dirt beneath the nails. The fingernails are clipped and retained. The toenails are also short.

A needle puncture mark is noted on the ventral left foot. An intravenous catheter inserts into the dorsal right foot. All EKG pads and other medical therapy have been removed prior to the autopsy.

Except for the evidence of injury to be described, the remainder of the external examination is unremarkable.

Evidence of Injury

External Injuries: Multiple contusions and abrasions involve the head, trunk and extremities. Along the right forehead and right cheek is an 11.7 cm x 3.5 cm maroon contusion. On the lower right eyelid is a blue-pink contusion. On the upper mid forehead is a 2.0 cm faint pink contusion and on the lower left forehead is a 1.8 cm maroon contusion and a 0.1 cm red abrasion. On the bridge of the nose are red abrasions measuring up to 0.2 cm. On the tip of the nose, nares and nasal septum are multiple red and crusted abrasions measuring between 0.4 and 1.0 cm. Along the lateral left eyebrow is a 0.8 x 0.6 cm blue contusion and just lateral is a 0.8 cm maroon-blue contusion. On the left face is a 3.5 cm maroon contusion and 0.4 cm red abrasion. On the lateral left face to just below the left ear are 5 red and crusted abrasions measuring between 0.2 and 0.8 cm. On the helix of both ears are pink-purple contusions measuring 2.2 cm and 2.5 cm with crusted red abrasions on the right. Behind the left earlobe is a 0.3 cm healing crusted red abrasion and behind the right ear is a 0.2 cm red abrasion. On the lower right face is a 1.8 cm maroon contusion. On the right jaw are maroon contusions and red abrasions measuring between 1.2 cm and 2.0 cm. Just above the left upper lip is a 1 mm crusted red abrasion. Other small red crusted abrasions are also noted on the lower left face measuring between 0.1 and 0.3 cm. The lips are dry and on the left lower lip is a 0.4 cm red abrasion. The upper frenulum is disrupted and shows evidence of remote trauma with scar tissue present. There are also 2 small blue contusions on the inner upper lip. There is no other trauma noted oral cavity and no foreign material is noted in the mouth. The tongue is atraumatic.

On the posterior neck are several crusted red abrasions measuring between 0.2 and 0.3 cm. On the left lateral neck is a 2.5 cm linear red abrasion and a 0.2 cm red abrasion. On the nape of the neck/right back is a 0.8 cm maroon contusion.

Multiple contusions are present on the chest and abdomen. On the upper right anterolateral and anterior chest are maroon contusions measuring between 0.3 and 1.3 cm. On the lateral right chest are four maroon contusions measuring between 2.8 and 4.5 cm. On the mid chest are maroon contusions measuring up to 3.2 cm. Adjacent to the left nipple is a 1.5 cm maroon-pink contusion. Other maroon and maroon-blue contusions are on the upper, lower and lateral left chest measuring between 0.8 and 2 cm. On the right upper abdomen is a 1.2 cm blue contusion and on the lower right abdomen is a 3.5 cm blue to blue-green contusion. In the right inguinal region is a 6 x 3.5 cm maroon contusion. On the left upper abdomen is a 1 cm maroon-blue contusion and on the lower left abdomen is a 1 cm blue contusion. Overlying the mid and left pelvis is a 4 x 2 cm dark maroon to blue contusion. On the anterolateral to lateral left abdomen are scattered larger blue to blue-green contusions in aggregate measuring 6.8 cm.

On the upper right back, just below the right shoulder, is an oval maroon contusion with red dry abrasions measures 5 cm in greatest dimensions and consistent with a bite mark. Within this bite mark are linear and punctate red abrasions resembling teeth marks with the bite mark measuring approximately 3 cm across. A second bite mark is also on the right back and consists of upper and lower dark maroon to blue curvilinear contusions and dry red crusted abrasions resembling teeth marks measuring approximately 3.2 cm across superiorly and 2.8 cm across inferiorly. Extending from this bite mark is a 3.2 cm linear red abrasion with a second 0.4 cm red abrasion on the lateral right back. On the right shoulder are three maroon contusions measuring up to 1 cm. On the left upper back are multiple maroon to maroon-brown contusions measuring between 0.5 and 2.8 cm. On the mid back are maroon to light blue contusions measuring between 0.8 and 1.5 cm. On the mid to lower back are larger, darker maroon to blue contusions measuring between 1.5 and 3 cm.

The anal opening is unremarkable. A previous genital examination has been performed with a 1 mm red abrasion now present on the right labia minora and linear red abrasion on the left labia minora measuring 2 mm. On the left vestibule is a linear 4 mm red abrasion. The hymen is annular and there is no trauma noted. There is no trauma noted to the vaginal canal as well. A small thread of cotton-like material is noted within the vaginal canal, most likely related to the previous examination prior to the autopsy. No examination kit is obtained at autopsy since swabs and smears of the oral cavity, anus and vagina were collected prior to autopsy.

The upper and lower extremities have multiple contusions. On the left upper arm is a 3.5 cm maroon to blue-green contusion with four small red abrasions suspicious for a bite mark. On the posterior left arm is a dark maroon contusion measuring 10 x 4 cm. Other scattered maroon contusions extend along the posterior left arm to the left elbow measuring between 1.5 and 2 cm. Multiple maroon contusions extend down the posterior left forearm measuring 10 cm in aggregate. On the ventral left forearm are scattered crusted red abrasions measuring between 0.8 and 1.4 cm. On the medial and lateral left wrist are pink contusions measuring 1.0 and 1.4 cm. On the dorsal left hand and left third finger are maroon contusions measuring 1.0 and 0.3 cm. On the posterior and posterolateral right arm to the elbow are numerous dark maroon almost confluent contusions. On the right elbow are small red abrasions

measuring up to 0.6 cm. On the dorsal right forearm are multiple blue to maroon contusions measuring from 0.4 to 3.5 cm. On the ventral right forearm are scattered maroon contusions measuring between 0.4 and 0.8 cm, as well as a 3.5 cm blue contusion.

Along the lateral right hip is a maroon contusion measures 6.3 cm. A large area of blue to blue-maroon contusions extends along the lateral right thigh measuring 10.5 cm in aggregate. On the upper anterior right thigh are several small maroon contusions measuring up to 2.5 cm and a 5.8 x 3.5 cm maroon contusion. On the medial upper right thigh are two contusions, one blue and one brown, measuring 0.6 cm and 0.8 cm. Coalescing maroon to blue contusions are present along the medial right thigh measuring 4.5 cm in aggregate. On the lower right thigh is a maroon 2.5 cm contusion. On the anteromedial right knee are 3 maroon contusions measuring in aggregate 2.5 x 2 cm and on the posterolateral right knee is a 4 cm maroon contusion. Three long slender maroon contusions extend across the lateral right calf measuring 3.5 x 1 cm, 3.5 x 0.8 cm and 3.2 x 0.8 cm and approximately 1.5 cm apart and are suspicious for finger marks. On the ventral right calf is a 1.5 cm green contusion. Scattered maroon contusions are noted on the posterior right thigh and right calf measuring between 1 and 1.8 cm. On the anterior left thigh is a patterned contusion consisting of four long slender blue-green contusions measuring (from superior to inferior) 4.5 x 1.2 cm, 3 x 0.8 cm, 5.5 x 1.5 cm and 4.5 x 2.8 cm, spaced approximately 1 cm apart, and again, suspicious for finger marks. On the lower left thigh is a 6 x 2 cm dark maroon to blue contusion. On the medial left knee is a V-shaped dark maroon to brown contusion measuring 3.5 x 0.6 cm superiorly and 2.5 x 0.5 cm inferiorly. These 2 contusions most likely occurred from a blow with the knee bent (leg flexed). Several dark maroon contusions involve the anterior lower left thigh and left knee measuring 9 x 6.5 cm in aggregate. Also on the medial left knee are five small crusted red abrasions measuring between 0.1 cm and 0.3 cm. Multiple maroon to blue contusions involve the ventral left calf measuring 10 x 3.8 cm in aggregate. On the medial left heel are multiple dark maroon contusions measuring between 0.4 and 2 cm. Scattered faint pink to light blue contusions are noted on the dorsal left foot measuring up to 5 cm. Nailbed hemorrhage involves the right great toe with a blue contusion measuring 1.2 cm. On the dorsal right foot is a 2 cm blue contusion. On the plantar aspect of the right foot is a 5 x 1.8 cm pink to blue contusion with a central 0. 5 cm crusted superficial laceration.

As previously mentioned the skin turgor is poor and the eyes are sunken consistent with dehydration.

Internal Injuries: Subsequent autopsy of the head reveals multiple areas of scalp contusions involving the left frontal, temporal and parietal scalp, the right parietal scalp and the occipital scalp and measuring between 0.2 cm and 10 cm. On opening the cranial vault, subdural hemorrhage overlies both cerebral hemispheres superiorly and inferiorly (right > left) and cerebellum measuring approximately 25 cc with no gross evidence of organization. Subarachnoid hemorrhage is patchy and covers the inferior temporal lobes (right > left), the cerebellum and brainstem. The brain is sent to neuropathologist Stephen J. Nelson for consultation with the gross examination under internal examination (head, brain and spinal cord). The eyes are removed and there is bilateral perineural hemorrhage noted. The eyes are sent for an eye pathology consultation with the Eye Pathology Laboratory (please see separate report). The spinal cord is removed with subdural hemorrhage noted, especially in the cervical spinal canal, measuring < 5 cc. Patchy subdural hemorrhage is noted throughout the thoracic spinal canal. No gross injuries are noted to the spinal cord.

Subsequent autopsy of the chest reveals small contusions involving the posterolateral aspects of the right lower lobe and the apex of the right upper lobe of the right lung. Contusions involve the left lung, lower lobe, posteriorly and posterolaterally.

Subsequent autopsy of the abdomen reveals a faint contusion involving the upper pole of the right kidney measuring 2.5 cm.

The back is incised and many of the contusions already noted on the back have dermal and subcutaneous hemorrhage and rarely have hemorrhage extending into the musculature of the back. Subcutaneous hemorrhage (contusion) is noted within the right buttock and not noted on the skin surface.

The left humerus is removed and a linear diagonal (spiral) fracture is noted and confirms the fracture noted on x-ray.

Internal Examination:

Head, brain and spinal cord: The scalp is incised and retracted. No skull fractures are found. The cranial vault is opened. The epidural space is free of hemorrhage. The dura is thin, tough and pliable. The brain in a fresh state weighs 1110 grams. The cerebral hemispheres, mid brain and pons appear normally developed. The cerebral vessels at the base of the brain also appear normally developed and there are no aneurysms or AV malformations seen. The brain is forwarded for examination by neuropathologist Stephen Nelson with the follow description (please see the neuropathology report):

Portions of the included dura mater contain thin films of non-adherent subdural blood. The dural venous sinuses are patent on serial cross sectioning. There is no meningeal staining or discoloration.

The fixed brain weighs 1,050 grams. According to your autopsy report, the brain weight at autopsy was 1,110 grams. There is no gross evidence of a defect in segmentation and/or prosencephalic cleavage, neurulation, or in segmentation and/or prosencephalic cleavage. There is no defect in cortical development. The leptomeninges are largely translucent. Focal subarachnoid hemorrhage is present on the inferior surface of the temporal lobes and parasagittal, anterior to the central sulcus of Rolando. There is an oblique post-mortem rent in the inferior surface of the left frontal lobe extending from the interhemispheric fissure to the left temporal pole. The cranial nerves are grossly unremarkable. The arterial circle of Willis at the base of the brain is that of a normal "adult" configuration. There is no evidence of significant atherosclerosis, vascular anomaly or aneurysmal dilatation. The pituitary and pineal glands are grossly unremarkable. There is some grooving of the right uncinate process that is associated with softening and discoloration. There is no subfalcine (cingulate) herniation.

The infratentorial structures are externally remarkable for side-to-side midline shift of the midbrain with entrapment of the cerebral peduncle. The leptomeninges are, nonetheless, essentially clear and without significant subarachnoid hemorrhage or discoloration.

Sequential coronal sections of the cerebrum, cut from temporal poles to occipital poles reveal slightly ventriculomegaly extending anterior to the temporal poles. There is no midline shift. The lateral ventricles are without compression or displacement. The foramen of Monro and the aqueduct of Sylvius are patent. The choroid plexus and ependyma are intact and grossly unremarkable; no xanthogranulomata are within the lateral ventricles. The cortical gray ribbon is intact throughout, except for the inferior surface of the left frontal pole (as previously described externally) where an artifactual handling artifact was present. The subjacent cerebral white matter is unremarkable grossly and it is soft and gelatinous and the tinctorial properties are somewhat pink and consistent with incomplete penetration of the formalin fixative to this depth. The basal ganglia, thalami and hippocampal formations are bilaterally symmetric. The mammillary bodies are preserved. The septum pellucidum is not cavum at the level of the genu or the splenium. The corpus callosum appears artifactually severed in the interhemispheric fissure throughout its length, from genu to splenium.

Sequential horizontal sections of the midbrain, pons, cerebellum and medulla oblongata are cut at right angles to the neuroaxis. Small petechial-like hemorrhages are seen on the cut surfaces of the cerebral peduncles. Otherwise, they are without significant gross abnormality. The pigmentation of both the locus ceruleus and substantia nigra is unapparent and consistent with the decedent's reported chronologic age. The cerebellum displays no unusual gross features. The deep midline nuclei of the cerebellum are symmetric and the folia are within normal limits. The medulla oblongata, including the pyramids, is symmetric.

A segment of spinal cord (61/2 inches long) was received previously serially cross sectioned at 5 mm intervals. The segment includes the thoracic and lumbar dilatations and a short portion of the cauda equina. The leptomeningeal blood vessels are diffusely congested.

Body: The body is opened with a Y-shaped incision and the back is incised. The organs occupy their usual positions and relationships. There are no significant fluid accumulations within the pleural, pericardial or peritoneal cavities.

Neck: The hyoid bone, thyroid cartilage and cervical spine are intact. A neck dissection is performed and there is no hemorrhage within the strap muscles of the neck. The thyroid gland is composed of tan, waxy soft tissue. There is no tracheoesophageal fistula.

Cardiovascular System: The heart weighs 52.3 grams and the epicardial, endocardial and pericardial surfaces are smooth and glistening. The coronary arteries follow the usual right dominant distribution and arise from 2 ostia. On cut sections, the myocardium is beefy red and firm. There are no atrioseptal or ventriculoseptal defects. The cardiac valves are thin, delicate and normally formed. The aorta and vessels arising from the aorta are normally developed. The venous return to the heart is normal.

Respiratory Tract: The right lung weighs 69.8 grams; the left lung weighs 54.9 grams. The pleural surfaces are pink-maroon and smooth and glistening. The injuries have been previously described. On cut sections, there is no evidence of infection or tumor. There are no pulmonary emboli. The trachea and bronchi are normally developed and there is no aspiration.

Liver and Biliary Tract: The liver weighs 280 grams and the capsule is smooth and glistening. On cut sections, the parenchyma is tan-red and soft. The gallbladder is present and contains thin green bile.

Gastrointestinal Tract: The esophagus is lined by the usual tan-gray wrinkled mucosa. The stomach, when opened contains less than 3 mL of green fluid. The small intestine is also opened and also shows just a small amount of green thick fluid with no food material noted. The large intestine contains green-yellow, soft fecal material. There is no obstruction noted. The mesentery is thin and transparent.

Adrenal Glands: Unremarkable.

Pancreas: Unremarkable.

Hematopoietic System: The spleen weighs 20 grams and the capsule is smooth and blue-gray. On cut sections, the parenchyma is maroon and soft. There is no generalized lymphadenopathy. The thymus is composed of tan, lobulated soft tissue. The bone marrow is red and apparently cellular.

Genitourinary Tract: The right kidney weighs 24.5 grams and the left kidney weighs 32.5 grams. The capsules strip with ease revealing smooth cortical surfaces. On cut sections, the corticomedullary demarcations are distinct and the parenchyma is tan-red and soft. The collecting systems and ureters are not dilated. The bladder is empty. The uterus, bilateral fallopian tubes and ovaries are appropriate for age with a small 2 mm cyst noted within the left ovary.

Miscellaneous:

Toxicology: Toxicology was performed on the postmortem blood at NMS Labs and reveals benzoylecgonine at less than 50 ng/mL as well as acetone and isopropanol. A vitreous electrolyte panel is performed on the left vitreous only and reveals a blood urea nitrogen level of 67 mg/dL, a creatinine of 1.4 mg/dL; a sodium of 159 mmol/l; a potassium of 12.6 mmol/l, a chloride of 167 mmol/l and a carbon dioxide of 3. These findings are consistent with dehydration.

X-rays – full body X-rays are taken and reveal the left humerus fracture removed. The fracture is consistent with a spiral fracture.

Fingernail clippings and clippers from both hands are released to the investigating agency.

Neuropathology and Eye Pathology consultations are performed on this case. Please see these separate reports.

Microscopic Description:

Brain and spinal cord: The following is the neuropathology microscopic description (please see the neuropathology report):

Eighteen (18) glass microscopic slides were prepared from paraffin histology blocks, and each was stained initially with hematoxylin & eosin (H&E).

The dura mater displays subdural and intradural acute hemorrhage. No significant numbers of inflammatory cells are identified microscopically. Scattered formalin pigment is also identified, but no hemosiderin pigment or pigment-laden macrophages are present. No microscopic evidence of organization of the subdural hemorrhage is present.

The neuronal laminations of the neocortex display the typical six layers. There is no histologic evidence of any disorder of neuronal proliferation or migration.

The leptomeninges contain extravasated intact erythrocytes. The erythrocytes display no poikilocytosis. Few neutrophils are identified marginated within the leptomeningeal blood vessels, but there is no diapedesis. No hemosiderin-laden macrophages are present in the subarachnoid space. The leptomeningeal blood vessels

are diffusely congested. The penetrating arteries and arterioles have some extravasated erythrocytes within the cortex and in the subcortical U-fibers of the superficial white matter at the gray-white junction. This is associated with endothelial cell swelling.

There is prominent vacuolation of the white matter, particularly within the interdigitating white matter near the cortex. A rare arteriole within the white matter contains a fibrin thrombus with marginated neutrophils. Small penetrating arterioles adjacent to the lateral geniculate nucleus contain perivascular hemosiderin-laden macrophages. The cortical neurons display widespread hypereosinophilic cytoplasm, nuclear pyknosis, and some karyorrhexis.

The hippocampus displays diffuse neuronal necrosis, including Sommer's sector (CA-1). The included ependyma and choroid plexus are histologically unremarkable.

Similar microscopic changes are present in the ipsilateral cerebral peduncle involving the pyramidal neurons of the pars compacta of the substantia nigra. No neuromelanin pigmentation is seen within those cells, consistent with chronologic age. The pontine tegmentum displays bilaterally symmetric neurons with widespread hypereosinophilic cytoplasm, nuclear pyknosis, and karyorrhexis. Portions of the tectum contain microscopic ependymal rests. The area postrema displays only vascular congestion.

The leptomeningeal blood vessels are congested. No significant extravasated erythrocytes are visualized outside of the blood vessels. No external granular lamina is present. The penetrating blood vessels contain minimal perivascular erythrocyte extravasation.

The spinal cord leptomeninges are acellular. There is diffuse vascular congestion. The central canal is widely patent throughout. Focal ependymal rests are identified at the level of Clarke's column. There is no neuronophagia or inflammatory cell infiltrates.

Eight (8) selected glass slides were subsequently stained with antiserum to amyloid precursor protein (APP). A positive control slide from brain was appropriately stained. No negative control slide was employed. These 8 sections included the cerebrum, brainstem (pons), and cerebellum. All of these slides show positive staining with antiserum to amyloid precursor protein in various neuroanatomic locations, but predominately in the white matter of the interdigitating white matter of the cerebrum, centrum semiovale, and in the midline of the basis pontis involving the longitudinal fiber pathways. The white matter, as previously described, demonstrates widespread diffuse edema with myelin swelling.

Right kidney: Sections show areas of intraparenchymal hemorrhage and congestion.

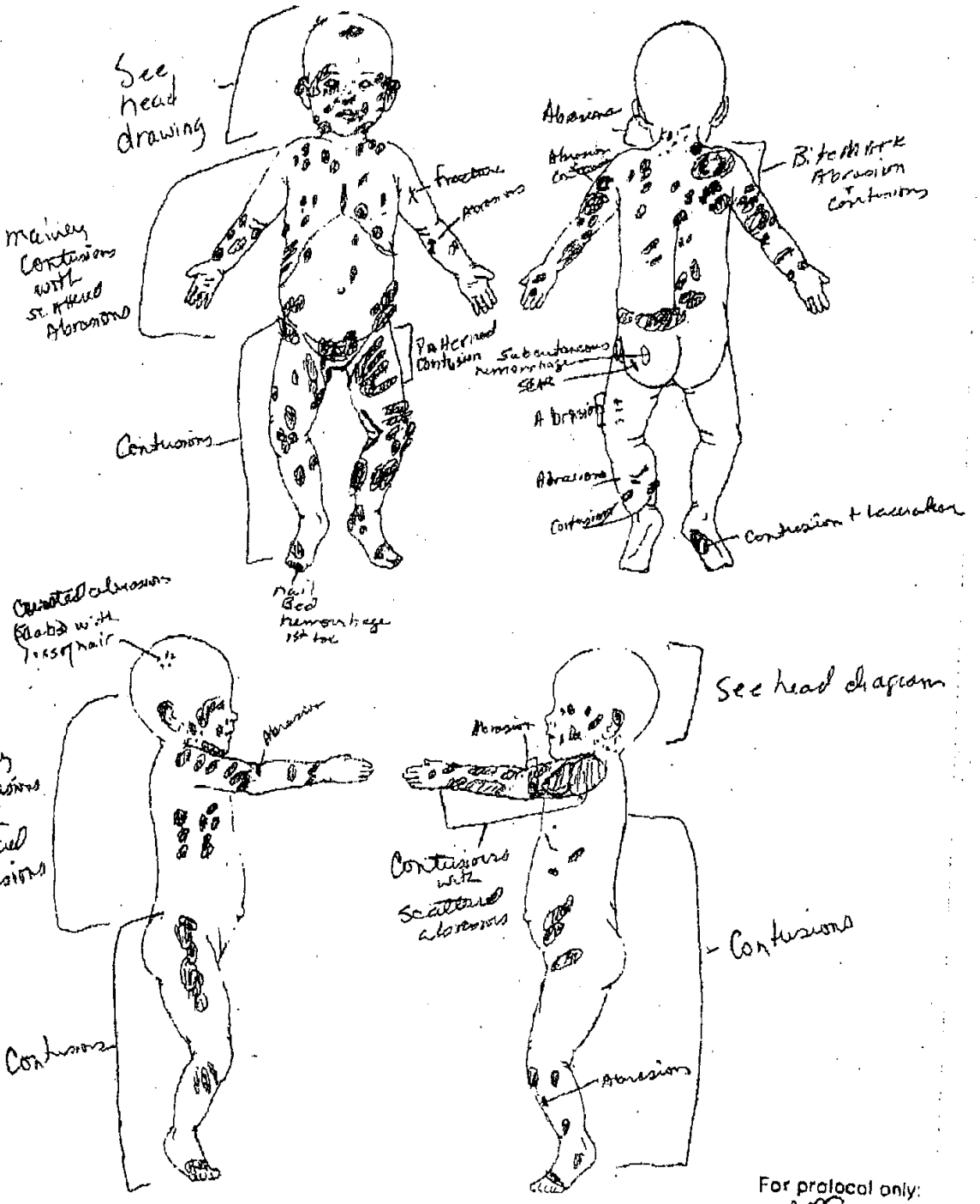
Lungs: Sections show pulmonary edema involving both lungs with pulmonary congestion, atelectasis and macrophages in alveolar spaces. There is intra-alveolar hemorrhage consistent with the contusions seen on gross examination. No acute inflammation is seen.

Left humerus: Sections show a healing fracture with bone marrow fibrosis, hemorrhage, granulation tissue and bone remodeling. New bone formations is prominent. No acute inflammation is seen.

Sections are taken of multiple contusions. Sections of the scalp contusions show mainly acute hemorrhage within the subcutaneous tissue without hemosiderin deposition. Other contusions of the abdomen, pelvis and chest show subcutaneous and dermal hemorrhage, most without pigmented histocytes.

Name Alvarez, Myriah Autopsy No. 0707-38

Color C/H Age 2 Sex F Date 2/18 + 2/19/07



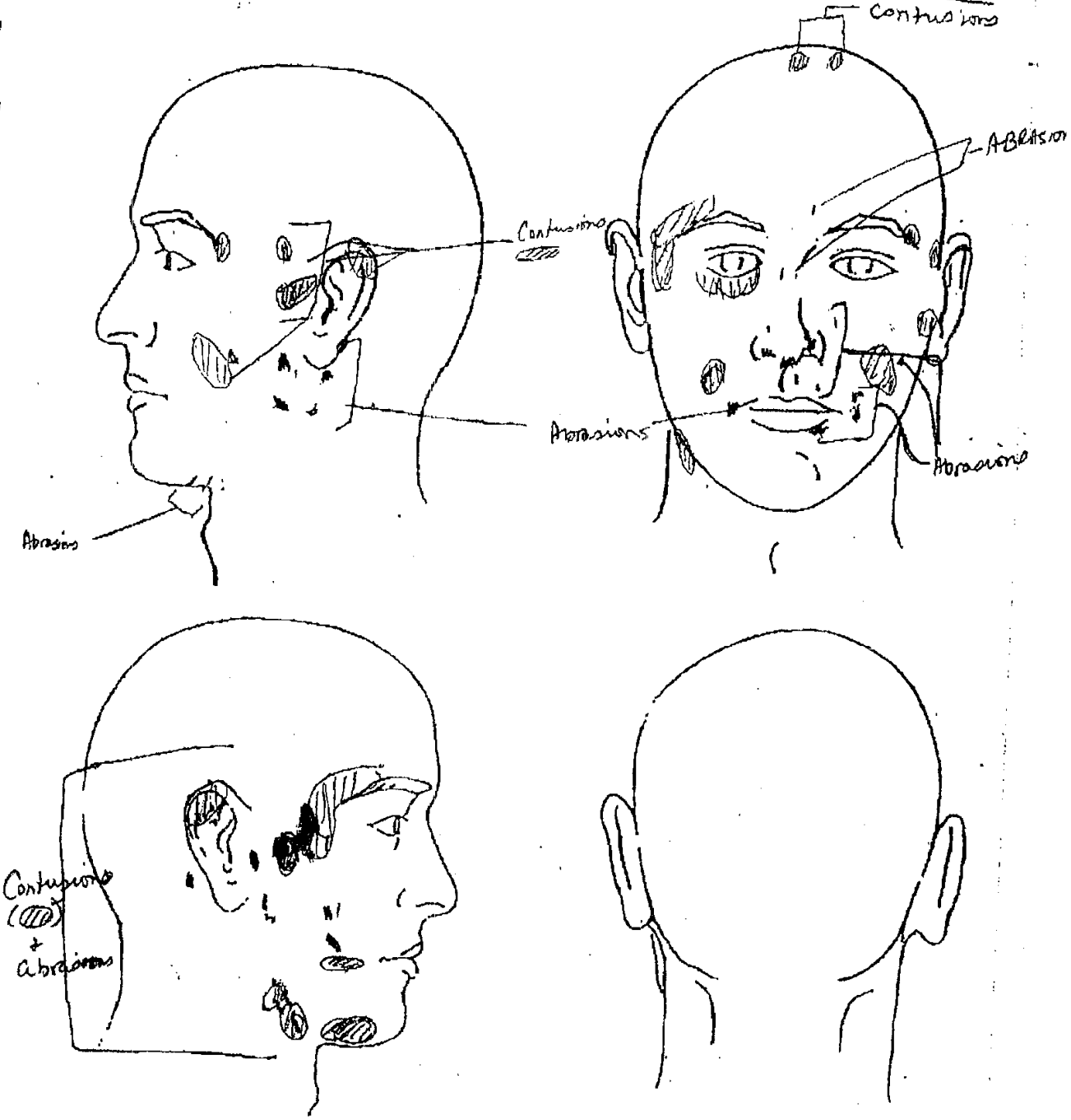
For protocol only:

ME FORM #5 1014

PAGE 32

Name Alvarez, Myriah Autopsy No. OA-07-38

Color C/H Age 2 Sex F Date 2/18 + 2/19/07



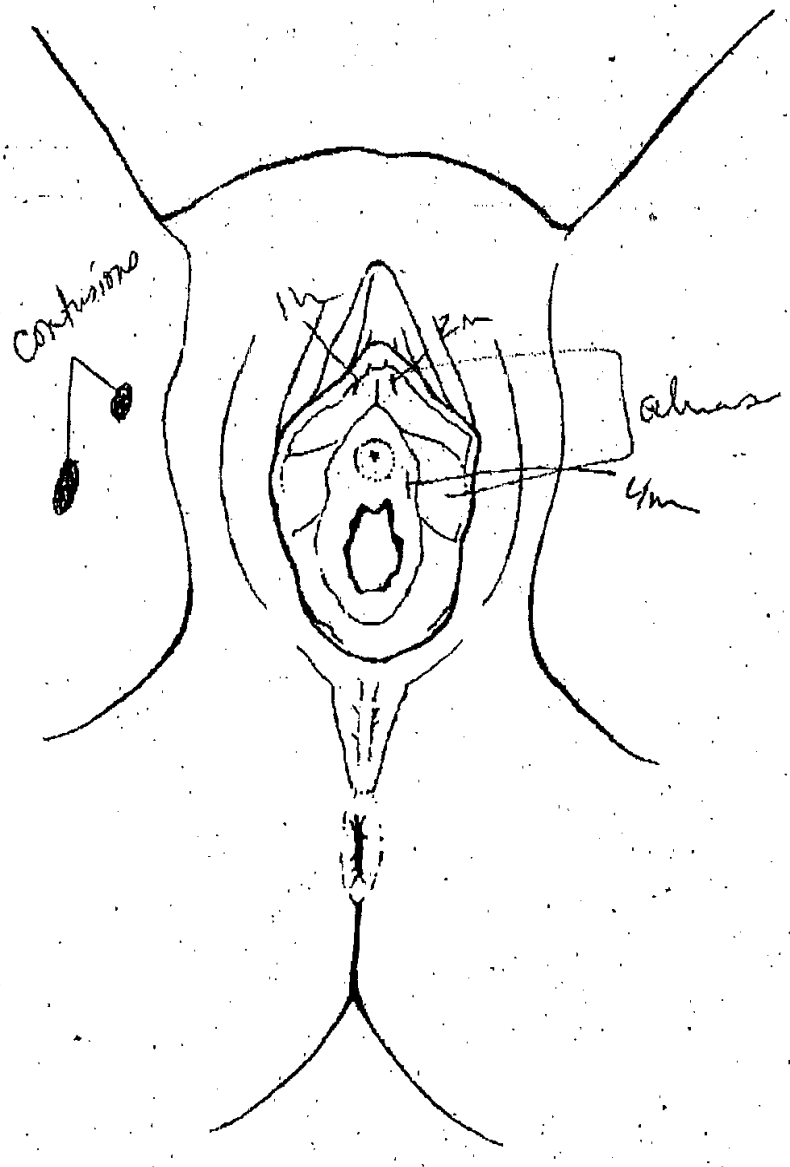
For protocol only:

NAF

Perineum, female

Name Alvarez, Myriah Autopsy No. A07-38

Age 2 Race C/H Sex F Date 2/14/07



8H 48

MAYO MEDICAL LABORATORIES
A Division within Mayo Reference Services
1-800-533-1710 OR 16H

C7026143
Tel : 956-389-4500

OA-07-38

Valley Baptist Med Center
Attn: Lab
PO Drawer 2588/2101 Pease Street
Harlingen, TX 78550

Accession # : A9924171
Patient Name : ALVAREZ,MYRIAH
Birth Date:09/16/2004 Age:2 Gend
Medical Rec #: MORGUE-5526;1
Client Acct #: M62029
Ordering Phys: FARLEY, NORMA
Collect Date : 02/19/2007 10:00 AM
Received Date: 02/22/2007 11:01 AM

Specimen Information : C7026143

Test Requested	Result	Units	Ref Range	Per Sit
THERAPUETIC DRUGS NMS				

Testing is complete. Results have been faxed and an attachment has been mailed.
Test Performed by:National Medical Services, Inc.
3701 Welsh Road
P.O. Box 433A
Willow Grove, PA 19090-0437

#Performing Site Legend

Patient Name : ALVAREZ,MYRIAH
Acct Status : FINAL REPORT

Report Printed : 03/14/2007 8:39 AM
Page : 1

NMS LABS
NMS Labs
3701 Welsh Road, PO Box 433A, Willow Grove, PA 19090-0437
Phone: (215) 657-4900 Fax: (215) 657-2972
e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, DABFT, DABCC, Laboratory Director

CONFIDENTIAL

March 8, 2007

TO: 60136
Mayo Medical New England
Attn: Mayo Medical Lab/N.E.
265 Ballard Vale Street
Wilmington, MA 01887

TOXICOLOGY REPORT OF: ALVAREZ, Myriah 2/F
NMS Workorder No: 07056711
Client ID No: A9924171

SPECIMENS: Two gray vials (one containing ~ 6 mL of blood and one containing ~ 5.5 mL of blood) were received on 02/23/07.

EXAMINATION: Analysis Requested - Panel 8102B - Autopsy Toxicology Therapeutic and Abused Drug Screen

FINDINGS:

Blood

ISOPROPANOL (by Headspace GC)	3.0 mg/dL
ACETONE (by Headspace GC)	4.0 mg/dL
COCAINE CROSS-REACTIVES (by ELISA)	Positive
COCAINE (by GC/MS)	None Detected Reporting Limit: 50 nanog/mL
COCAETHYLENE (by GC/MS)	None Detected Reporting Limit: 50 nanog/mL
BENZOYLECGONINE (by GC/MS)	Less than 50 nanog/mL
ATROPINE (by GC/MS)	Trace

Incidental findings by GC/MS: NICOTINE.

Other than the above findings, examination of the specimens submitted did not reveal any positive findings of toxicological significance by procedures outlined in the accompanying Analysis Summary.

CONFIDENTIAL

NMS Workorder No: 07056711
Client ID No: A9924171

COMMENTS:

1. Isopropyl alcohol (isopropanol, 2-propanol) is a common industrial and laboratory chemical that is available as a 70% aqueous solution in "Rubbing Alcohol." It is readily absorbed following vapor inhalation, dermal application, or oral administration, and is distributed into all body water (Volume of Distribution = 0.6 L/kg). In contrast to ethyl alcohol, isopropyl alcohol exhibits first order kinetics in man, with an elimination half-life between 2.5 and 3 hr. The acetone formed as a metabolite has an elimination half-life of about 22 hr.

   Isopropyl alcohol produces effects in man similar to those produced by ethyl alcohol (impairment of cognitive, perceptual and psychomotor capabilities manifest as decrements in alertness, judgment, perception, coordination, response time and sense of care and caution). However as a central nervous system depressant, isopropyl alcohol has about two times the potency of ethanol. Therefore, while the effects produced are similar, the blood concentrations of isopropyl alcohol required to produce those effects are roughly half as great.

   In a study of 31 isopropyl alcohol deaths, postmortem blood concentrations ranged from 10 to 250 mg/dL (mean, 140 mg/dL) and acetone blood concentrations ranged from 40 to 300 mg/dL (mean, 170 mg/dL).

   Isopropyl alcohol and acetone are sometimes used as components in preparations used for embalming. Isopropanol and acetone may sometimes be detected in postmortem blood specimens as the result of contamination by embalming materials.

2. Acetone is a solvent used for chemicals, paints, etc. It is, however, also a product of diabetic - and fasting-induced ketoacidosis as well as a metabolite following isopropyl alcohol ingestion. In high concentrations, acetone can have CNS-depressing effects.

   Reported normal endogenous acetone levels are up to 1 mg/dL. Levels associated with diabetic or fasting ketoacidosis range from 10 to 70 mg/dL. After exposure to 100 and 500 ppm acetone for 2 hr, reported blood levels peaked at 2 and 10 mg acetone/dL, respectively. A blood level of 250 mg/dL was reported in an individual who became lethargic following ingestion of acetone.

3. Cocaine is a DEA Schedule II controlled excitant-stimulant drug. Benzoylecgonine is an inactive transformation product which can be formed via chemical action both inside and outside the body. Cocaethylene is a cocaine congener that is believed to be the product of a cocaine-ethanol interaction. Methylecgonine and ethylecgonine are other metabolites of cocaine. Following the use of a fully excitant-stimulant intranasal dose of 2 mg/Kg (approximately 140 mg in a 155 lb adult), reported peak plasma concentrations of cocaine average ≈200 nanog/mL at 1 hr. Outside the body, lower cocaine and higher benzoylecgonine concentrations than actually exist at the time the blood specimen was drawn can result. Cocaine manifestations include restlessness, risk taking, excitement and aggression. A period of mental and physical fatigue follow the use of cocaine after the excitant-stimulant effects wear off.

   Tissue concentrations of cocaine in cocaine-related fatalities can vary according to dosage, route of administration, survival time and specimen storage conditions. Reported blood levels in such fatalities range from 900 to 21,000 nanog/mL (mean, 5300 nanog/mL).

4. Atropine is an anticholinergic alkaloid used in pre-anesthetic therapy to control airway secretions and as an antispasmodic to control gastrointestinal spasms. It is frequently used as an antidote in the treatment of anticholinesterase-type pesticides. It can be obtained naturally from deadly nightshade or jimson weed. Atropine is also used in resuscitative attempts.

   Toxic effects of atropine have considerable individual variation; however, at high doses, signs and symptoms include mydriasis, hot dry reddened skin, deliriums and hallucinations.

   In resuscitative failure, most of the administered drug remains confined to the intravascular injection pathway. Often the drug is still present in the postmortem blood collected from the heart sampled at autopsy.

CONFIDENTIAL

NMS Workorder No: 07056711
Client ID No: A9924171

Respectfully,

Laura M. Labay, Ph.D.
Forensic Toxicologist

LML/jra

This analysis was performed under chain of custody. The chain of custody documentation is on file at NMS Labs.

Unless alternate arrangements are made by you, the remainder of the submitted specimens will be discarded six (6) weeks from the date of this report; and generated data will be discarded five (5) years from the date of this report.

****** **** ANALYSIS SUMMARY **** ******

8102B - Therapeutic and Abused Drug Screen

Test No. 8102B – Drug Screen by Enzyme-Linked Immunosorbent Assay (ELISA) on Blood for: Amphetamine, Barbiturates, Benzodiazepines, Benzoylecgonine (Cocaine), Cannabinoids (Marihuana), Methamphetamine, Opiates and Phencyclidine (PCP); Headspace Gas Chromatography for Ethanol, Methanol, Acetone and Isopropyl Alcohol.

Test No. 8102B - Drug Screen II- Gas Chromatography and Gas Chromatography/Mass Spectrometry Analysis on Blood:

The following is a general list of compound classes included in the Gas Chromatographic screen. Other specific compounds outside these classes are also included. Please note that not all known compounds included in each specified class or heading are included. The detection of any particular compound is concentration-dependent. For a detailed list of all compounds included in this screen, please contact NMS Labs.

Analgesics (opioid and non-opioid), Anesthetics, Antiasthmatic Agents, Anticholinergic Agents, Anticonvulsant Agents, Antidepressants, Antiemetic Agents, Antihistamines, Antiparkinsonian Agents, Antipsychotic Agents, Antitussive Agents, Anxiolytics (Benzodiazepine and others), Cardiovascular Agents (non-digitalis), Hallucinogens, Hypnosedatives (Barbiturate and others), Muscle Relaxants, Non-Steroidal Anti-Inflammatory Agents (excluding Salicylate) and Stimulants (Amphetamine-like and others).

Test No. 8102B - Colorimetric Analysis on Blood for: Salicylates and Acetaminophen.

Test No. 5637B - Cocaine and Metabolites - Gas Chromatography/Mass Spectrometry on Blood for: Cocaine, Cocaethylene and Benzoylecgonine.

******* END OF REPORT *******

CLIA# 43D0693628
CAP# 2143304

EYE PATHOLOGY LABORATORY
Lions Sight Research Center
UNIVERSITY OF TEXAS HEALTH SCIENCE CENTER
7703 FLOYD CURL DRIVE, MC 6230
SAN ANTONIO, TX 78229-3900
(210) 567-8460 Fax: (210) 567-8413

E07-109
Name: Mytiah Alvarez
Case #: OA07-38
DOB: 09/06/04 Sex: F
Autopsy Date: 2/19/07
Lab Date: 3/21/07

Physician: Norma Farley, MD
2101 Pease Street
Harlingen, TX 78551-2538

Code: 88307 X 2

SPECIMEN(S) SUBMITTED: A) Globe, OD. B) Globe, OS.

CLINICAL HISTORY: 2 year old female who had been in foster care until October 2006 and then placed back in the mother's home. The child presented to Valley Baptist Medical Center Emergency Room unresponsive with obvious bruising to the body in different stages of healing. The patient had a left humerus fracture also. The patient had sustained blunt head trauma with subarachnoid and subdural hemorrhage, numerous contusions to the head and body and bite marks to the back.

PREOP DIAGNOSIS: Post mortem enucleations, right and left globe.

GROSS DESCRIPTION: Submitted and received in formalin in a container labeled "Alvarez OA07-38" were two containers labeled A&B.

Specimen A reveals an intact infant's right globe whose laterality was confirmed to be accurate. This eye had no obvious signs of previous surgery or congenital defects. The globe measured 20 x 21 x 20 mm. The cornea was clear and measured 11 x 10 mm. The iris was brown in color and the pupil is round at 3.5 mm. The optic nerve segment is 22 mm long and there is obvious brown perineural pigment consistent with hemorrhage. The globe is opened in the equatorial meridian to facilitate inspection of the posterior pole. On gross examination there were multiple fixed retinal folds. No obvious hemorrhage was seen. A cross section of the optic nerve was obtained and photographs were taken of the obvious perineural pigmentation. Representative sections from the anterior and posterior eyewall as well as the optic nerve are submitted for routine histologic processing. Wet tissue remains.

Specimen B reveals an intact infant's left globe which measures 20 x 21 x 17 mm. The laterality was confirmed by external examination of anatomic landmarks. This globe did not have obvious signs of previous surgery or congenital defects. The cornea was 10.5 x 10 mm and clear. The iris color was brown and the pupil was round at 1-2 mm. The optic nerve segment was 31 mm long and there were obvious perineural pigmentation consistent with hemorrhage present. This globe was opened in the equatorial meridian and on inspection the posterior pole was remarkable for diffuse fixed retinal folds. There were no obvious retinal hemorrhages seen. A cross section of the optic nerve was obtained and photographed. The optic nerve was surrounded by brown pigment. Representative sections of the anterior and posterior eyewall as well as the optic nerve are submitted for routine histologic processing. Wet tissue remains.

MICROSCOPIC DESCRIPTION: Microscopic examination of Specimen A reveals a generally unremarkable cornea and anterior chamber. Small foci of iris pigment is present on the anterior lens capsule. Posteriorly the retina does not contain obvious fresh intraretinal hemorrhage however there are fixed retinal folds and focal disruptions of the interior limiting membrane. The optic nerve is surrounded by fresh hemorrhage present both within the subarachnoid and subdural space.

Microscopic examination of Specimen B reveals a relatively normal anterior chamber and angle. There is iris pigment present on the anterior lens capsule. Posteriorly there are fixed retinal folds with interruption of the

207-109

internal limiting membrane. There is no frank hemorrhage present. The optic nerve contains perineural hemorrhages present within the subarachnoid and subdural space.

DIAGNOSIS: (Based on gross and microscopic examination only)

A&B) Bilateral globes, status post mortem bilateral enucleations:

TRAUMATIC IRIS PIGMENTATION OF ANTERIOR LENS CAPSULE OU

BILATERAL FIXED RETINAL FOLDS

BILATERAL DISRUPTION INTERNAL LIMITING MEMBRANE

BILATERAL PERINEURAL HEMORRHAGE. SEE COMMENT.

COMMENT/DISCUSSION: A constellation of distinctive gross and microscopic features in this case are highly suggestive for nonaccidental trauma. Specifically, the optic nerve and intraocular injuries appear to have been sustained in an acute fashion no more than a few days prior to the child's death. There are no gross or microscopic features present which indicate a congenital ocular anomaly, preexisting ocular disease, or accidental trauma. The finding of optic nerve sheath hemorrhages involving the Circle of Zinn have been well documented in the medical literature as manifestations of nonaccidental trauma.

Frank W. Scribbick, III, MD
Clinical Associate Professor
Director, Ophthalmic Pathology Laboratory

FWS:mm-Transcribed 4/04/07 at 4:02 pm

02/18/2007
16:30

VALLEY BAPTIST MEDICAL CENTER
DEPARTMENT OF LABORATORY MEDICINE

INTERIM REPORT
1 PAGE

NAME: ALVAREZ,MYRIAH
H# : MORGUE-5526
ACCT: 000000000
AGE : 29M SEX: F
D.O.B.: 09/16/2004

OA-07-38

*LOC: MORGUE
*DR : FARLEY, NORMA
2101 PEASE STREET
HARLINGEN, TX 78550

X27273 COLL: 02/18/2007 08:30 REC: 02/18/2007 09:10 PHYS: FARLEY, NORMA

FEMORAL BLOOD	AUTOPSY SPECIMEN TYPE DRAWN		[VB]
HEART BLOOD	AUTOPSY SPECIMEN TYPE DRAWN		[VB]
POOLED BLOOD	AUTOPSY SPECIMEN TYPE DRAWN		[VB]

[VB] = Performed at VBMC Lab, 2101 Pease St. Hgn,Tx 78550

X27615 COLL: 02/18/2007 12:00 REC: 02/18/2007 12:36 PHYS: FARLEY, NORMA

LEFT EYE VITREOUS

UREA NITROGEN, BLOOD	H 67 VITREOUS FLUID	[7-22]	MG/DL	[VB]
CREATININE	H 1.4 VITREOUS FLUID	[0.5-1.2]	MG/DL	[VB]
ELECTROLYTES				
SODIUM	H 159 VITREOUS FLUID	[136-145]	MMOL/L	[VB]
POTASSIUM	* 12.6 VITREOUS FLUID	[3.5-5.1]	MMOL/L	[VB]
CHLORIDE	H 167 VITREOUS FLUID	[100-112]	MMOL/L	[VB]
CARBON DIOXIDE	* 3 VITREOUS FLUID	[22-27]	MMOL/L	[VB]

[VB] = Performed at VBMC Lab, 2101 Pease St. Hgn,Tx 78550

X27625 COLL: 02/18/2007 12:00 REC: 02/18/2007 12:38 PHYS: FARLEY, NORMA

UREA NITROGEN, BLOOD	REQUEST CREDITED DUPLICATE REQUEST SEE X17615	
CREATININE	REQUEST CREDITED	

ALVAREZ,MYRIAH
02/18/2007 16:30

CONTINUED

TAB 14

THOMAS G. ALLEN, PH.D.

19479 C.R. 1321 -- Flint, TX 75762
Phone: (903)894-9182 -- Fax: (903)894-6158 -- Email: thomasgallen@embarqmailcom

PERSONAL DATA

Date of Birth: February 6, 1948
Marital Status: Married Children: Two

EDUCATIONAL BACKGROUND

Ph.D. in Psychology & Minor in Statistics – Texas A&M University @Commerce, Commerce, TX, August 1984. Dissertation: "The Effects of Creativity and Personality on Time Estimates During Hemisphere Appropriate Task."

M.S. in General Experimental Psychology -- Texas A & M. University, College Station, TX, August 1975. Thesis: "Internal, Powerful Others, and Chance Locus of Control as Related to Performance in a Concept Identification Task."

B.A. in Psychology & double minors in Economics and Sociology -- Western New Mexico University, Silver City, NM., May 1970.

PROFESSIONAL EXPERIENCE

1995 -1996:	Institutional Review Board Meeting, East TX Medical Center Hospital (ETMC), P.O. Box 6400, Tyler, TX 75711.
1989 -1996:	Director, Preoperative Evaluation Program ETMC.
1987 -1990:	Consulting Psychologist, Rehabilitation Unit; Member, Rehabilitation Committee, ETMC.
1986 - 1995:	Consulting Psychologist, Pain Management Program, ETMC.
1985 - 2008:	Private Practice.
1984 -1985:	Director of Clinical Services, University Park Hospital, Tyler, TX
1983 -1984:	Program Director, Psychiatric Unit, Mother Frances Hospital, Tyler, TX.
1976 -1979:	Staff Clinical Psychologist, Admissions and Diagnostic Unit, Rusk State Hospital (RSH), Rusk, TX.
1975 -1976:	Unit Director, RSH.
1975 -1974:	Staff Psychologist, Maximum Security Unit, RSH.
1974:	Treatment Coordinator, Day Treatment Center, RHS.

COLLEGIATE AND ADJUNCT ACTIVITIES

1996:	Adjunct Professor, University of Texas at Tyler, Department of Psychology, Tyler, TX. Class taught: Theories of Personality
1995:	Adjunct Professor, University of Texas at Tyler, Department of Psychology, Tyler, TX. Class taught: Social Psychology
1985-1986:	Adjunct Professor, University of Texas at Tyler, Department of Psychology, Tyler, TX.

GRADUATE SCHOOL

1983 -1982:	Teaching Assistant, East TX State University, Commerce, TX
1978:	Attended Southwest Psychological Association (Spring)
1977:	Attended Workshop conducted by TX Department of Mental Health concerning ethical/legal issues with use of behavioral methods (Fall)
1977:	Member, Behavior Modification Review Committee, Rusk State Hospital,

Rusk, TX.
1974: Texas A & M University, Brazos County MHMR (Spring)
1973: Research Assistant, TX A & M University, College Station, TX
1973: Texas A & M University/Brazos County MHMR

UNDERGRADUATE SCHOOL

Member of Blue Key Honors Fraternity
Board of Directors, Pi Gamma Mu Honor Society
Member of University Student Senate
President of Senior Class
Secretary of Circle K International Service Club
Rotarian Student of the Year
Member of Psi Chi National Honor Society in Psychology

MEMBERSHIPS

American Psychological Association
APA Division of Psychology and Law
TX Psychological Association
East TX Psychological Association, 1995 President

CONTINUING EDUCATION

2009: American Academy of Forensic Psychology: Assessment of Competence to Stand Trial. May 24, 2009
American Academy of Forensic Psychology: Introduction to Assessment of Psychopathy Using the Hare Scales. May 23, 2009.
American Academy of Forensic Psychology: Forensic Report Writing. May 22, 2009.
2008: Texas Psychological Association: 11/20/2008 – Ethical and Practical Issues Using Psychological Testing in Forensic Assessment; 11/20/2008 – The Psychological Expert, Preparing for Court; 11/21/2008 – Apathy Dysphoria, and Cognition in elderly; 11/22/2008 Dealing with Subpoenas;
2007: Psychologists as Members of Healthcare Teams: Tools for Assessment, Interventions and Consultation with Medical Patients and Physicians. Illinois Psychological Association. November 10, 2007.
2007: MMPI-2: Updates for Clinical, Consulting, and Forensic Practice. Paul Arbisi, Ph.D., A.B.P.P. Illinois Psychological Association. November 8, 2007
2007: Applied Ethics and Law for Texas Psychologists. Texas Psychological Association.
2006: Mental Health in the Courtroom, October 25-28th. TX Department of State Health Service; Regional and Local Services, Continuing Education, 1100 West 49th Street, Austin, TX 75756.
2005: Competency to Proceed and Not Guilty By Reason of Insanity: Beyond the Basics, Sponsored by American Academy of Forensic Psychology. February 13, 2005, In Dallas, TX. Workshop Leader Kenneth H. Smail, Ph.D. and Chair of AAFP CE Program Alan M. Goldstein, Ph.D.
2005: Comprehensive Assessment of Malingering in Forensic Settings, Sponsored by American Academy of Forensic Psychology. February 11, 2005, in Dallas, TX. Workshop Leader, Richard Frederick, Ph.D. and Chair of AAFP CE Program Alan M. Goldstein, Ph.D.
2005: Introduction to Professional Liability and Risk Management: Module I. January

10, 2005. Sponsored by American Psychological Association, Cynthia D. Belar, Ph.D. Executive Director, Education Directorate.

2004: Sex Offenders and Victims, Presented by Anna Salter, Ph.D. Sponsored by Specialized Training Services, Inc., December 9-10, 2004 in Austin, TX.

2003: Evaluating Competency to Stand Trial. Workshop Presented for the Texas Psychological Association Annual Convention in Dallas, TX, November 7th,

2003: Presented by Mary Alice Conroy, Ph.D., ABPP (forensic) Sam Houston State University.

2003: Use of Problem Based Learning: The A, B, C, of Ethics, Step by Step. Ray Hays, Ph.D., J. D. University of Texas, Houston Medical School, Department of Psychiatry and Behavioral Science. Workshop Presented for the Texas Psychological Association, Annual Convention, Dallas, TX, November 7th.

2003: Dreams: Explore the Power of the Dreaming World. Presented by Franklin A. Shaffer, Ed.D, R.N. Sponsored by Cross County University. Tyler, TX.

2003: Advanced Forensic Psychological Practice: Issues and Application, Presented by American Academy of Forensic Psychology, Three days, Dallas, TX. Topics covered: Juvenile Justice Issues; Malingering Assessment of Response Styles; Criminal: Miranda Waivers, Mens Rea, Capital Cases; Personal Injury Damages and Liability; Testimonial Jurisprudence; Child Custody & Termination of Parental Rights; Testamentary Competence; Employment Discrimination, Sexual Harassment, ADA Sex Offender Risk Assessment; Violence Risk Assessment.

2002: Personality Assessment with the MMPI-2, Presented by Richard Lewak, Ph.D. of Specialized Training Services, Inc., in Dallas, TX.

2001: Risk Assessment of Sexual Offenders, Presented by Amy Phenix with Specialized Training Services, Inc., Arlington, TX.

2000: Violence Risk and Threat Assessment; Presented by Specialized Training Services, Inc., Austin, TX.

1999: Soulfood Psychotherapy; Presented by Noreen Vickrey, Ph.D., Sponsored by East Texas Psychological Association.

1999: Accelerated Hypnotherapy Course; Presented by American Institute of Hypnotherapy; Little Rock, Arkansas.

1998: Protecting the Professional; Presented by Daryl Green; Sponsored by Institute for Personal Growth and Achievement, Inc., Ft. Worth, TX.

1998: Risk Management in the Evolving Health Care Market. Presented by Eric A. Harris, Ed.D, J.D.; Sponsored by American Psychological Associate Insurance Trust and the East Texas Psychological Association.

1998: Psychological Practice in Long Term Care Facilities. Presented by Alan Stephenson, Ph.D.; Sponsored by East Texas Psychological Association.

1997: Childhood Memory aka *Jeopardy in the Courtroom*. Presented by Stephen J. Ceci, Ph.D. Continuing Professional Education. University of Mary Hardin Baylor, PO Box 8002, UMHB Station, Belton, TX 76513

1996: Current Development in Practice Management. Presented by Mr. Bill Brown. Health Care Consultant, East Texas Psychological Association.

1996: Improving Ethical Decision Making. Presented by Michael Gottlieb, Ph.D. East Texas Psychological Association.

1996 Child Sexual Abuse Evaluation: Research, Assessment Issues and Legal Application Presented by Michael P. Maloney, Ph.D. American Academy of Forensic Psychology.

1996: Competency to Stand Trial and Criminal Responsibility Assessment. Presented by Kathleen P. Stafford, Ph.D. American Academy of Forensic Psychology.

1996: Preparing for the Diplomat Exam in Forensic Psychology. Presented by Curtis L. Barrett, Ph.D. American Academy of Forensic Psychology.

1996: The Role of the Psychologist in Death Penalty Litigation. Presented by Sandra B. McPherson, Ph.D. American Academy of Forensic Psychology.
1995: Treating Psychological Trauma in Children. Presented by Karen Sitterle, Ph.D.
1995: Psychology Practice Ethics and the Courts. Presented by Judge Larry Gist, and Curtis Wills, Ed.D, J.D..
1995: Political and Legislative Actions Affecting the Practice of Psychology in Texas. Presented by Thomas Kozak, Ph.D., Legislative Liaison of the Texas Psychological Association.
1994: Forensic Evaluations and Forensic Applications of the MMPI and MMPI-2. Presented by Stuart Greenberg, Ph.D., and Kevin Moreland, Ph.D.
1993: The National Mediation Academy, Inc.
1990: American Academy of Pain Management.
1989: The Institute for Somatic Psychotherapy.
1986: Applied Stress Research Foundation.
1985: Clinical Applications of the MMPI. Presented by James Butcher, Ph.D., University of Minnesota.

PROFESSIONAL PAPERS

Allen, T. G. and Fournet, G. "The Effects of Creativity and Personality on Time Estimates During Hemisphere Appropriate Tasks", Presented to the Thirtieth Annual Convention of the Southwestern Psychological Association New Orleans, LA. April, 1984.

Gary L. Huber, M.D., Brigid B. Dorman, Ph.D., Ed.D., Thomas G. Allen, Ph.D. and Robert J. Pandina, Ph.D. "The Role of Nicotine in Smoking Behavior." 1995.

CERTIFICATION

Certified as Mediator and Neutral in compliance with provisions of Section 154.02 of Texas Civil Practices and Remedies Code.

LICENSURE

Licensed Psychologist #2955, Texas State Board Examiners of Psychologists

REFERENCES

Will be given upon request.

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June 19, 2010
CONFIDENTIAL

Ms. Margaret Schmucker
Attorney at Law
512 E. 11th Street, Suite 205
Austin, TX 78701

Re: ***Psychological Consultation*** *in Appeal No. AP 76.020 Melissa Lucio. In the 138th District Court of Cameron County, Texas.*

Dear Ms. Schmucker:

Per your request I examined the above named defendant on May 19, 2010 in the Texas Department of Criminal Justice death row unit in Gatesville, Texas. My direct forensic examination lasted 3 hours. The purpose of the examination as well as the limits of confidentiality were explained to the defendant, I read a statement regarding same to the examinee, and inquired as to whether or not she had questions about the statement. The examinee indicated she had no questions, and signed a document indicating she understood my explanation, and chose to proceed with the examination. I conducted a forensic interview, took some history from the examinee, and administered the Personality Assessment Inventory (PAI) and the Minnesota Multiphasic Personality Inventory-II-RF to the defendant. In addition to the above I studied records provided that were case related to include offense report, statements by the examinee, 2 prior psychological evaluations and legal material submitted by her attorneys. Case data included a long, videotaped interrogation. In addition to interview, review of archival material and current psychological testing, Historical, Clinical and Risk factors as outlined in the HCR-20 were used to assess risk for violence. I assessed Psychopathy using the Hare Psychopathy Checklist-Revised (PCL-R).

BACKGROUND INFORMATION AND INITIAL OBSERVATIONS

The examinee is a 40 year old Hispanic American born 6/18/1969 in Lubbock, Texas. She was raised mostly in Harlingen and Houston. She completed grade 10 in Harlingen high school and has never obtained a GED or any additional, formal education. Her primary language is English. Her mother is Esperanza Trevino who lives in Harlingen and the examinee reports a good relationship with her. Her biological father is Santos Gonzales and she maintains no relationship with him as he has been in and out of her life. She reports a good relationship with a step-father, Olegario Trevino, who currently lives in Laguna Vista, Texas. He and her mother are divorced, but they were married when Mrs. Lucio was age 8. She has 2 sisters and 3 brothers. The examinee quit school at age 16 due to pregnancy apparently and married her first husband. The examinee has described a family life during her developmental years as chaotic,

impoverished and including family violence. Despite that she recalls being outgoing and happy as a child.

During this contact the examinee appeared to be genuinely cooperative. There was no clinical indication of evasiveness or efforts to feign cognitive or psychiatric disorder.

Psychiatric History

The defendant reports no psychiatric history. She has undergone some counseling related to CPS intervention in 2004 that included parenting classes and psychological evaluation dated November 4, 2004 by Xavier Martinez, Ph.D. A risk assessment dated July 5, 2008 was performed John Pinkerman, Ph.D. subsequent to the start of her trial for Capital Murder June 30, 2008, but she was actually seen prior to the trial.

Education

As noted above the examinee indicates she completed grade 10 and has no additional education. She did not like school but reports no particular conflicts with teachers or authority figures in general. There is no indication of any pattern of behavior problems, to include indications of aggression or anti-sociality during the developmental years. She self-describes as an average student.

Medications

The examinee reports she is currently prescribe Trazadone 200 mg. h.s. for sleep.

Family and Marital History

The examinee indicates she became pregnant at age 16 and married Guadalupe Lucio in 1985. They were separated in August 1994 and never obtained an official divorce. That union resulted in 5 children: Daniela, age 23; Melissa, age 21; John, age 20, Alexandra, age 18; Selena, age 17. Daniela is now married and lives in Laguna Vista with 4 children. Melissa is single with 3 children and lives in Harlingen. John is single and lives in Harlingen. Alexandra is single with one child and lives in Harlingen. Selena is single and still in high school but living with a boyfriend.

Mrs. Lucio has been in a common-law marriage to Roberto Alvarez since 1994. They have had 8 children; Rene is age 13, Richard is age 12, Robert is age 19, Gabriel is age 9, Adriana is age 8, Sarah is age 7, and Erin and Adrian are twins age 2 ½. Roberto Alvarez was jailed in 2003 and Mrs. Lucio had an affair with David Diaz and Mariah, the victim in the Capital Murder, was a product of that affair.

As noted above the examinee's developmental years were marked by family chaos and poverty. It does not appear she ever really bonded with her biological father, but did appear to have developed parental relationships with mother and step-father. At least one brother has had substance abuse problems.

Criminal History

The examinee had a DWI in 2006. CPS was contacted by the hospital on two

occasions because the examinee tested positive for cocaine either during pregnancy or at the time of the births of the children, Gabriel and Sarah who are now ages 10 and 7. Mrs. Lucio's children were removed for about 2 months. The issue at the time of CPS intervention was essentially child neglect. There was no allegation of physical abuse.

Substance Use

The examinee has tested positive for cocaine on 2 occasions. She reports her first use of cocaine was around age 16 or 17, use was intermittent and she discontinued use when her children were removed in 2006.

Employment

The examinee has a spotty work history and this is understandable with so many children. When she was employed it was mostly doing cashier work at restaurants, auto parts stores and convenience stores. Her last job was in home health care where she worked for about 6-8 months.

Mental Status Exam

The examinee presents with a normal limits mental status. There are no hallucinations or delusions. Mood is depressed but she is coping well in a very difficult situation. Affect remains full range but is mildly blunted, most likely associated with an ongoing grief reaction and depression. She becomes tearful when providing her account of the death of Mariah. Attention and concentration are intact and no impairment in short-term or long-term memory is apparent. She was able to recall 6 digits forward and 5 digits backward, which is normal limits. She shows good speech articulation and there is no indication of receptive or expressive aphasia. Thoughts are rational, goal oriented and responses are relevant to questions asked. Sleep impairment is present, but the Trazadone helps. She continues to have some difficulty with sleep onset and there is some middle awakening. Personality functioning appears normal limits and she is a very pleasant person although she could be characterized as naïve. There is no indication of antisociality, narcissism or borderline personality development. There is no history of head injury or seizure disorder.

Version of Events

The examinee has provided her version of events on prior occasions although I did review with her the conduct for which she was convicted, both her version and the State's version. I will not repeat those versions here other than to indicate the State's position was that she murdered her child, had been abusive to the child while Mrs. Lucio denies such abuse and indicated the child died after falling down stairs and she did not seek medical help in a timely fashion.

Psychological Testing

On the PAI the examinee provided a valid profile. Her response pattern did not indicate Positive Impression Management and there was mild disparity between the Negative Impression Management and Positive Impression Management scales, but not at a level that invalidated test results. The disparity probably represents a cry for help. Significant distress is indicated by a single scale elevation of the Depression Scale.

There was an upper normal limits elevation on the Substance Abuse Scale, consistent with the history of the examinee for cocaine abuse. Persons with similar profiles are experiencing severe unhappiness and pessimism. She is plagued by thoughts of worthlessness, failure and hopelessness. Anergia and sleep impairment are likely a part of the clinical picture. Further, no problems with empathy, hostility or paranoia were identified. There appear to be no difficulties with unusual thoughts or odd experiences or over activation. Mrs. Lucio's self-concept is suffering. She negatively appraises herself, is prone to be self critical and pessimistic. Scales assessing aggression were low.

On the MMPI-II-RF the examinee provided a valid profile. Emotional-internalizing findings were present, likely explaining a mild elevation on over-reporting, which probably accurately reflects clinical distress. No psychotic disorder was identified. The examinee is likely to be experiencing severe distress expressed in somatic and cognitive complaints involving vague neurological complaints and head pain. She is likely prone to develop physical symptoms in response to stress. Emotional dysfunction is present as indicated by suicidal ideation. Her current situation is difficult and she is experiencing sadness and unhappiness. She is significantly depressed with frequent rumination, self-doubt and inefficaciousness. She is likely passive-dependent. She is reporting significant anxiety likely to be accompanied by anxiety related disorders, intrusive ideation and sleep problems. She is suspicious of others in terms of ideas of persecution as she feels overwhelmed and believes others seek to harm her. No issues related to aggression were identified on the MMPI-II-RF.

Prior Psychological Testing

The most recent testing by Dr. Pinkerman in 2008 showed the examinee to be functioning in the low-normal range of intelligence using the WAIS-III full scale test of intelligence. The MMPI-2 did not show malingering, but did show considerable distress along with dependent personality features. Antisocial Personality Disorder was not identified. No psychotic disorder was identified.

Assessment of Symptom Validity

Symptom validity was not an issue with this examinee. There was no effort to appear mentally ill or to feign cognitive impairment. The examinee's approach to clinical evaluation appeared forthright.

Risk Assessment

Using the HCR-20 to systematically assess risk factors that have been identified in the literature as relevant to risk assessment the examinee would be placed in the low risk category. The factors assessed are listed below:

Historical

The examinee's history of previous violence is pretty slim. The case record suggests she had bitten the child victim on one occasion and CPS intervened for child neglect, but these are not considered instrumental violence and do not reflect an identifiable pattern of violence upon which to place the examinee in a high risk category of risk for future violence. The evidence at trial that the examinee abused her children

lacked credibility for me. The child neglect issue was relevant, but the neglect had more to do with impoverishment than direct violence as typically seen in violent offenders.

Clinical interview and the record do not indicate any pattern of violence beginning at a young age. There are no juvenile adjudications or contacts with juvenile authorities.

The examinee has been in 2 long-term, committed relationships. She shows no pattern of many short term relationships and no pattern of absent relationships.

The examinee has worked and has a spotty work history, but her main work has been as a homemaker with a lot of children, though she and the family lived in poverty.

The examinee does have a history that includes the use of cocaine and one DWI. There was no indication that her substance use was related to the death of the victim. She also apparently responded to intervention when her children were removed by CPS and I believe the DWI has been adjudicated with no additional events.

The examinee shows an absence of major mental illness. There was no indication of such an illness identified in current or prior psychological evaluations and no indication especially of those illnesses most often associated with acts of violence such as paranoid schizophrenia.

Prior and current psychological evaluations do not indicate the examinee is a psychopath and same is not indicated in other case related records. Despite her impoverished and chaotic upbringing the examinee did not develop a pattern of maladjustment during the developmental years and does not suffer from Antisocial Personality Disorder, or Borderline Personality Disorder, both of which are disorders most often associated with violence.

The examinee appeared to respond to intervention by CPS, at least within the limits defined by her poverty.

Clinical

The examinee does not suffer from a major mental illness; she is not psychotic. Prior and current psychological evaluations do not identify problems with insight. Further, prior evaluations do not indicate she holds negative attitudes that foster or justify violence. There is no mental illness that energizes violence or potential for violence. The fact that she has used cocaine and has one DWI could be used as an indication of impulsivity, but this would be considered a weak predictor of risk for violence.

Risk Management

The examinee is most likely to respond to plans for remediation and is likely to willingly participate in such planning. There are no clinical risk factors to deal with such as mental illness, and she has already shown historical responsiveness to intervention for use of cocaine. Her current adjustment to incarceration is non-problematic in that, as far

as I can determine, she has no infractions and no violent incidences.

Diagnostics

Axis I: Major Depression
Axis II: None

Conclusions

As you know I am not a judge or a jury and have no opinion about guilt or innocence and am allowed no such opinion by law. This is a consultation report and I am happy to discuss the case with you. I must express some concerns as a part of my consultation role and if this report is used at any trial it may have to be redacted, but I'll leave that to the attorneys and the court.

Mrs. Lucio's current clinical examination and testing indicate significant stress with accompanying anxiety and depression. She tends to internalize conflicts and stress and is prone to develop stress related disorders. She is not diagnosable with a psychotic disorder. Given her circumstance on death row her distress is understandable, and she continues to grieve not only her situation, but the loss of her child and her family. There is really little, if any, indication that this woman represents a risk for continuing acts of violence either in or out of prison. I do not know how this issue was addressed at trial, but to classify her as a risk for continuing acts of violence is not supported by the research literature and frankly the notion approaches the preposterous. Violence is actually considered a rare event, making assessment of potential risk difficult and it is commonly over predicted. To assess risk for violence requires looking at multiple dimensions, but no one factor can be utilized in singular fashion. The context of violence potential must also be taken into account. If it was reasoned that she was a risk to small children for example, then the fact that there are no small children in prison should have been taken into account. In the field of criminal risk assessment the most problematic arena is the instrumental violence of the typical violent offender in which violence is used to obtain a criminal goal. Other violence is impulsive and often a product of drug or alcohol use. Still other violence is purely situation based in which only if certain, specific situational factors are present will the violence occur. In cases of serial violence the prediction is actually easier because of the compulsive nature of the violence, such as serial sex offenders, and serial killers. In this case there is a profound lack of evidence clinically and psychometrically to indicate Mrs. Lucio represents a risk for continuing acts of violence. I must also point out that when CPC intervened there was no indication at that time of physical abuse directed at her children. The only issue was poverty resulting in neglect.

If I may be of further service please do not hesitate to contact me.

Sincerely,

Thomas G. Allen, Ph.D.
Psychologist

State of Texas
County of Smith
Sworn to and subscribed before me on the 3rd day of November, 2010.
Andrea Renea Bivonet
Notary Publics Signature
My Commission Expires 2-16-11

ANDREA [illegible] Notary Public [illegible] February 16, 2011

TAB 15

Affidavit of
Dr. John Pinkerman, PhD.
State of Texas
County of Cameron

Before me, the undersigned notary in and for the Cameron County within the state of Texas, on September 22, 2010 personally appeared John Pinkerman, PhD who after being by me duly sworn, depose and says:

My name is Dr. John Pinkerman, Ph.D. I am a Texas licensed clinical psychologist with a general practice in the Rio Grande Valley. As part of my practice I have provided forensic psychological services in civil and criminal matters before federal as well as state courts. On December 10, 2007 I received an appointment as a defense psychologist with the law firm of Peter C. Gilman. Mr. Gilman was the lead defense attorney for Melissa E. Lucio. Melissa Lucio was going to trial for capital murder involving the death of her two and one half year-old daughter, Mariah. Mr. Adolfo Cordova was the second chair counsel assisting Mr. Gilman. Ms. Norma Villanueva, a licensed clinical social worker and I were appointed to assist defense counsel in preparation for the trial. I expected to participate in multiple meetings regarding Ms. Lucio's prior history, current psychological status and the development of mitigating evidence in the event she was convicted.

To the best of my recollection, the defense team met on two occasions. I also remember making several calls to Mr. Gilman and Mr. Cordova. In preparation for participation in the defense team I did independent review of research on issues related to false confessions and domestic violence.

Prior to her trial I reviewed all of the available records related to Mrs. Lucio's involvement with the state of Texas Department of Child protective services, videotapes of her confession to police, autopsy photographs of Mariah Alvarez and Mrs. Lucio's offense list. In the process of conducting my assessment and arriving at my professional opinions I met with Mrs. Lucio on four occasions while she was detained waiting trial. The purpose of these interviews was to obtain her social history and conduct psychological tests. Those tests included the Mini-Mental Status Exam, the sentence completion blank, the Wechsler Adult Intelligence Scale-III, the Minnesota Multiphasic Personality Inventory-2 and the Millon Clinical Multiaxial Inventory-III.

I recall having serious questions about the nature of Mrs. Lucio's interrogation and confession. In my professional opinion her psychological characteristics increase the likelihood she would acquiesce while providing her confession. My review of her videotaped statements revealed that she was isolated for approximately 5 hours, repeatedly interrogated by male police officers in close quarters, was not provided a place or opportunity to rest nor provided food or water. As I recall this interrogation went on until late in the night. During meetings with defense counsel I raised questions about these issues. To my knowledge these issues were never raised at the pretrial or trial. I was prepared to testify regarding research related to false confessions and Ms. Lucio's

psychological characteristics which increase the likelihood of false confession. Ms. Lucio's behavior during her interrogation by the police could have been accounted for by her dependent and acquiescent personality. Her history had ample instances of self-sacrifice combined with emotional and physically abusive relationships with males. She appeared to take the most gratification from her role as a mother despite being overwhelmed by those responsibilities. She appeared very capable of making self-sacrifice in providing a false confession in order to avoid investigation of her children.

In addition, early in the development of this case I had questions and reservations about her culpability in the death of her daughter Mariah. The family's history indicated that several children had behavioral disorders marked by severe aggression against the siblings. Prior medical records as well as CPS records suggest that there were bite marks on the children while the children were in foster care before Mariah's death. The historical record offered little support for the idea that Ms. Lucio physically abused her children. Mariah's bruises were extensive and the children were described as very physical with each other. I also raised questions about the physical evidence pertaining to bite marks on Mariah's body. I respectfully asked if dental experts were called in for consultation. I felt that these factors; sibling abuse, prior bite marks and a dental expert were not developed nor offered in the guilt/innocence phase of the trial. If I had been called by defense counsel as a witness I worry would've offered my general opinions about sibling abuse and the characteristics often associated with mothers who kill their children.

During our meetings, I discussed the use of psychological findings in combination with Ms. Villarreal's use of social history has mitigating factors in the event Ms. Lucio was convicted of capital murder. Despite having been appraised of the wealth of information available for this purpose, trial counsel did not develop this as evidence or fully present it during the punishment phase of Ms. Lucio's trial although I was called during the punishment phase to testify on the issue of future dangerousness, my testimony was brief and not elaborated. It was my impression that defense counsel failed to explore several avenues open as a result of my psychological evaluation. These matters had been discussed prior to my testimony but were not developed.

I would have testified that Ms. Lucio displays poor verbal comprehension skills and her personality organization was characterized by repression and denial and disassociation. I would have stated that disassociation is defined as the isolation of thoughts from feelings and would account for much of her presentation recorded in the videotape of her confession. These processes of disassociation served to keep important elements and feelings away from conscious life. Individuals using disassociation often numb their feelings and appear empty or passive. It was evident from early childhood Ms. Lucio is subject to emotional physical and sexual abuse. Despite opportunities for intervention either with her as a child or as the mother her children, effective interventions were not made to break the vicious spiral downward into more capacity and dependency. She learned that her outcries were ineffective and was better to cultivate a predictable and secure abusive relationship than risk losing her family and identity as a mother.

Ms. Lucio's personality characteristics in combination with circumstances of Mariah's death were not congruent with fatal violence against her child. Ms. Lucio failed to meet the criteria for many of the prior identified subtypes of child murder mothers.

In her interrogation, Ms. Lucio denies having fatally injured Mariah. Only when she was approached with sympathy and understanding about how tragic her daughter's death was, did she begin to feel emotions and acquiesce to the confession. She consistently denied having used excessive physical discipline with their children. Several hours after Mariah's death, she said; "I'm responsible for it". She appears to be taking responsibility for the whole configuration of the abuse and medical neglect by the family, leading to her daughter's death but she does not admit striking Mariah in the head.

If called as a witness during a state or federal review hearing, I would also testify that during our meetings Ms. Villanueva and myself raised questions about a contract therapist from Child Protective Services being sent into the jail to conduct "therapy sessions" with Mrs. Lucio while she was under indictment for capital murder. We both felt this was an unusual procedure and appeared to put the mental health professional in conflict with the dual role. To my knowledge trial counsel failed to take any action on this issue and did not raise it during the trial.

In my professional opinion the limited number of meetings between me and other defense team members were insufficient to integrate our professional work and assist in a viable and available defense in either the guilt/innocence or in the punishment phase. My impression was that Mr. Cordova was asking follow-up questions but his hands were indirectly being tied by Mr. Gilman. In our meetings and while providing testimony it was clear that mental health issues were not being fully developed or addressed. It was unusual, based on my past experience in capital cases, to not be used more effectively.

In addition, I felt there were a number of avenues that should have been developed by defense counsel regarding the mitigating factors for Ms. Lucio. On one occasion I attempted to provide an extended answer in order to provide sufficient explanation of Ms. Lucio's personality dynamics and future risk factors. My effort was unsuccessful.

John Pinkerman, PhD. 12-3-10

State of Texas
County of Cameron

Denise Marie Toldo

DENISE MARIE TOLDO
Notary Public
State of Texas
My Commission Expires
May 09, 2011

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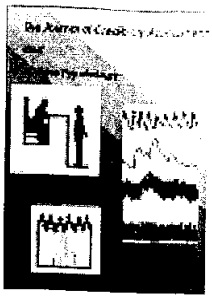

The Psychology of False Confessions

Richard P. Conti[1]

ABSTRACT: Obtaining a confession is one of the most important aims of police interrogation, and it is estimated that more than 80% of solved criminal cases are solved by a confession. However, a significant number of confessions that result in wrongful convictions are obtained through coercive questioning. This paper examines false confessions and discusses the psychological and social factors that influence innocent suspects to give self-incriminating false statements during police interrogation. Inherently coercive police questioning techniques that are employed to obtain confessions from suspects in-custody are presented.

The Psychology of False Confessions

Introduction

Frequently regarded as the most unequivocal evidence of guilt, a confes sion relieves doubts in the minds of judges and jurors more than any other evidence (Driver, 1968; Kassin & Wrightsman, 1985; Reik, 1959; Schafer, 1968; Wrightsman, Nietzel, & Fortune, 1994). In criminal law, the confes sion evidence is considered to be the most damaging form of evidence pro duced at a trial (Underwager & Wakefield, 1992; Wrightsman & Kassin, 1993; Zimbardo, 1967) and a prosecutor's most potent weapon (Kassin & Sukel, 1997) -- so potent that, in the words of one legal scholar, "the introduction of a confession makes the other aspects of a trial in court superfluous, and the real trial, for all practical purposes, occurs when the confession is obtained" (McCormick, 1972, p. 316). Confession evidence alone generally ensures a

[1] Richard P. Conti received his MA in Forensic Psychology from the John Jay College of Criminal Justice. He is currently completing the requirements for a Ph. D. in Clinical Psychology at the Fielding Institute where is concurrently enrolled in the Neuropsychology Certificate Program. The author thanks Melanie A. Kirk of the College of Saint Elizabeth for her editorial suggestions and helpful comments on earlier drafts of this article. Please address correspondence to: Richard P. Conti, Psychology Department College of Saint Elizabeth, Morristown, NJ 07960-6989.

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conviction (Driver, 1968; Kassin & Wrightsman, 1985; Schafer, 1968; Wrightsman et al., 1994). To obtain a confession is one of the most important aims of police interrogation (Underwager & Wakefield, 1992). Zimbardo (1967) estimated that of those criminal cases that are solved, more than 80% are solved by a confession. Dr. W. Sargant (cited in Brandon & Davies, 1973), speaking at the 5th Scientific Congress of the British Academy of Forensic Sciences, estimated that without confessions, convictions might be reduced by more than 70%. J. Barry of the Australian Supreme Court, addressing the United Nations, stated that "confession is the most attractive way of solving crimes" (cited in Brandon & Davies, 1973).

Confessions are a very powerful form of evidence. This may be due to observers misattributing the cause of the confession as being internal to the person (e.g., actual guilt) while discounting situational factors (e.g., possible coercion) which may not be readily apparent to an observer (Gilbert & Malone, 1995). In social psychology, this is known as the fundamental attribution error: the tendency to attribute other people's behavior to more dispositional (internal) causes and underestimate the importance of situational (external) factors (Ross, 1977). As Wrightsman (1991) points out, "It seems that what you say is more influential than why you say it" (p. 170). Understanding the fundamental attribution error may help explain how people, especially jurors, can be influenced by confession evidence even if the confession is considered unreliable. Once a suspect makes a confession, even if the confession is ruled inadmissible by the court, people often hold on to newly formed beliefs even after they have been discredited (Anderson, Lepper, & Ross, 1980) or instructed to ignore them by a judge (Kassin & Sukel, 1997; Kassin, Williams, & Saunders, 1990).

Are all confessions authentic? Do all suspected individuals give true confessions out of their own volition, devoid of duress during police interrogation? Bedau and Radelet (1987) revealed that the primary cause for the conviction of 49 (11.4%) of the 350 instances of miscarriages of justice in the U. S. this century was a false confession generated by coercive questioning. Bedau and Radelet operationally define a miscarriage of justice as "those cases which: (a) The defendant was convicted of homicide or sentenced to death for rape; and (b) when either (i) no such crime actually occurred, or (ii) the defendant was legally and physically uninvolved in the crime." (p. 45). In 309 (88%) of the cases innocence was established by state decisions indicating error (e.g., reversal by trial or appellate court).

How often do false confessions lead to miscarriages of justice? Wrightsman and Kassin (1993) report that no one knows but cited Lloyd-Bostock's report (1989) that in Great Britain, false confessions ranked second only to mistaken identifications as a cause of wrongful conviction among cases referred to the Court of Appeal. How often false confessions result in wrongful convictions is obscure (Gudjonsson & MacKeith, 1988; Kassin & Fong, 1999, Leo, 1998) although some observers (Gudjonsson, 1992; Kassin & Wrightsman, 1985; Leo, 1998; Leo & Ofshe, 1998; White, 1998; Wrightsman

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& Kassin, 1993; Wrightsman et al., 1994; Zimbardo, 1967) affirm that enough cases have been documented to suggest that a concern over such a risk is justified. Leo (1998) points out three reasons why it is impossible to even es timate the incidence or prevalence of false confessions: (1) police interroga tions are conducted in secrecy and they are usually not recorded, (2) law en forcement agencies do not keep records on the number of interrogations con ducted, and (3) it is difficult to establish what actually occurred to elicit a confession, especially if the confession resulted in a conviction.

As Leo (1998) points out, it is very difficult to establish a baseline on the incidence or prevalence of false confessions in the United States. Huff, Rattner, and Sagarin (1986) gave a conservative estimate of 6,000 wrongful convictions in the United States for index crimes[2] alone. The authors dis tributed surveys asking respondents who were directly involved in the crimi nal justice system (e.g., judges) to give estimates on the frequency of wrongful convictions. Using 1981 data for index crimes, Huff et al. used a 50% conviction rate and a wrongful conviction rate of only one-half of 1% (which was lower than most respondents estimated) to arrive at their esti mate. The authors concluded that even if a system is 99.5 % accurate, a high-volume area could produce 6,000 erroneous convictions per year. Keep ing in mind the data from the Bedau and Radelet (1987) study which showed that 11.4% of the convictions in their sample were the result of a false con fession, one could make a conservative estimate of 5% and still show that 300 false confessions result each year in a high-volume area.

The primary purpose of this paper is to examine the reasons why inno cent people sometimes confess. To be more specific, what factors compel innocent suspects to give false statements and confess to crimes they did not commit during police questioning? Secondly, how do interrogators get suspects to confess; what psychological and social influence do they employ? Although there are a limited number of research studies regarding false confessions, the present review of the literature indicates a shift in court rulings throughout United States history with regard to the attainment of confessions and the admissibility and validity of confession evidence. Differ ent perspectives on false confessions will be analyzed. Three psychologically distinct types of false confessions--voluntary, coerced-compliant, and co erced-internalized (Kassin, 1997, 1998; Kassin & Sukel, 1997; Kassin & Wrightsman, 1985; Wrightsman & Kassin, 1993)--with the two latter types being significant with regard to police interrogation will be explored. In addi tion, some related case histories including landmark decisions such as *Miranda v. Arizona* (1966) and *Brown v. Mississippi* (1936) are reviewed. Em phasis is placed on the psychological perspectives of coerced-internalized false confessions and other factors that influence false confessions. De mand characteristics of the police interrogation process are also be dis cussed, and several types of psychological and social factors employed by po

[2] According to the Federal Bureau of Investigation (1997) the following are considered to be index crimes: Homicide, rape, robbery, assault, burglary, larceny, motor vehicle theft, and arson.

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lice interrogators in order to secure confessions from suspects are presented.

A Historical Overview of False Confessions

Four centuries ago, a confession was treated as a conviction. The use of physical torture to extract confessions was common, and all confessions were routinely admitted into evidence without question. But slowly over the centuries, the status of confessions in the legal system shifted from the courts' limiting the admissibility into evidence of ordinary confessions in the mid-1700s, to totally excluding coerced confessions by the mid to late 1800s. By the 19th century, the courts were cynical of all confessions and tended to dismiss them if questionable.

In the early 1900s, U. S. courts were increasingly faced with cases of Black defendants who were said to have "confessed" to crimes after being physically beaten by the police (Kassin, 1997; Kassin & Wrightsman, 1985; Wrightsman & Kassin, 1993; Wrightsman et al., 1994). The case of *Brown v. Mississippi* (1936) was a landmark decision on this matter. In that case, three Black men were apprehended by the police for murder. The men were not allowed to consult with an attorney and were subsequently threatened, beaten, and tortured. Each of the three men eventually signed a police written "confession" to the murder. Each defendant was convicted and then sentenced to death. The Supreme Court of the United States reversed the convictions in *Brown* on the grounds that the police had violated the defendants' rights to due process of law. The Court ruled that evidence procured through physical torture and brutality must be excluded from trials. Furthermore, the Court asserted that a trial "is a mere pretense where the state authorities have continued a conviction resting solely upon confessions obtained by violence" (p. 287). Thus, the admissibility of confession evidence is prefaced by the requirement that the confession be proved voluntary (Kassin, 1997; Kassin & Wrightsman, 1985). That is, the confession must be given freely and knowingly (Gudjonsson, 1992; White, 1998), without physical or psychological coercion (McCormick, 1972, 1992), and in an unconstrained manner by the individual (*Culombe v. Connecticut,* 1961). The ruling in *Brown* set a precedent "that a state court conviction resting upon a confession extorted by brutality and violence violated the accused's general right to due process guaranteed by the Fourteenth Amendment" (McCormick, 1992, p. 232).

In determining the admissibility of confession evidence, the courts have considered other factors such as mental abuse in addition to physical force and threats. In the case of *Chambers v. Florida* (1940), the Supreme Court ruled that five days of prolonged questioning and other factors that fell just short of physical violence elicited concerns that the confessions given by the defendants were in danger of being false. An investigation into the totality of the circumstances surrounding the confessions was required as in the case of *Haynes v. Washington,* (1963). The defendant was refused telephone contact with his family and attorney and was told by police that these

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requests might be granted as soon as he made a statement. The Supreme Court of the United States ruled that the defendant's confessions were coerced by the fact that the defendant's "will" was overborne in an "atmosphere of substantial coercion and inducement created by statements and acts of state authorities" (p. 513). In the landmark case *Miranda v. Arizona* (1966), the Supreme Court ruled that unless the accused is advised by the police of his constitutional rights to remain silent and to obtain counsel, all self-incriminating statements are inadmissible in court.

To measure the accuracy of confession evidence, one would have to assess the combined frequency with which truly guilty people confess and truly innocent people do not. Thus, two types of false errors are possible: false negatives, in which guilty suspects fail to confess, and false positives, in which suspects who are innocent confess (Kassin, 1997; Kassin & Wrightsman, 1985). According to Kassin (1997), the false positive error, although less common than the false negative, poses a more serious dilemma for the courts. Kassin (1997) stressed the importance of knowing what factors increase the risk of a false confession.

As noted earlier, a 1987 study by Bedau and Radelet discovered 350 instances of miscarriages of justice in the United States alone. In each of these cases an innocent individual was convicted of murder or rape. In 49 of these cases, the foremost reason for the conviction was a false confession brought about by coercive interrogation. For several years, a coerced confession that lead to a conviction resulted in an automatic reversal of the conviction; however, this was changed in the case of *Arizona v. Fulminante* (1991). In this case, the Supreme Court of the United States ruled that a conviction based on a coerced confession was not to be automatically reversed. If the prosecution could prove beyond a reasonable doubt that the trial court error was harmless, the "harmless error" rule would apply. That is, other sufficient evidence must exist to sustain the conviction.

A Psychoanalytical Perspective on False Confessions

Theodor Reik (1959), renowned psychoanalyst and criminologist, state a belief that false statements originate from the unconscious compulsive need to confess. If instinctual impulses striving for expression are spurned or condemned by the external world, the still feeble ego can manage only to express them in the form of confession. Hence, the inclination to confess is a modified urge for the expression of the drives. Reik asserted that the unconscious compulsion to confess gratifies the need for punishment. That is, the need for punishment shifts from punishment to confession. He expounds further in his book, translated in 1959, *The Compulsion to Confess:*

Compare the situation with that of a little boy who seems to fear punishment for some secret misdeed. . . . least of all does he fear the punishment itself. Rather, he shows feeling of anxiety because of what his parents may be thinking as they learn of his little misdeed and because he must

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confess it to them. He has transformed the fear of punishment into the fear of confession. The confession itself, as that which precedes the punishment, has now become in the highest degree terrifying. The child himself says in many cases that it is not punishment he fears, but the scene in which he will tell his parents what he did. (pp. 202-203)

Here, the need for punishment, like any other strong drive, emits severe stress and pressure. The intensity of these impulses can be lessened only by partial gratification (Reik, 1959; Schafer, 1968).

Procedural Considerations

Opponents of questionable and inappropriate police interrogation and in vestigative procedures argue that a false confession is nothing more than the product of police incompetence (Ofshe, 1991; Zimbardo, 1967) and police viciousness. According to this view, in attempting to elicit confessions from suspects, police interrogators may use outright lies and subtler forms of de ception (Underwager & Wakefield, 1992; Wood, 1995). The most widely used and influential textbook on police interrogations (Gudjonsson, 1992, 1994; Huff, Rattner, & Sagarin, 1996), Inbau, Reid, and Buckley's (1986) *Criminal In terrogations and Confessions* encourages and describes a step-by-step process by which interrogators are to elicit a confession from a suspect by using out right deceit and psychological manipulation. Inbau et al. recommend telling suspects that they have evidence linking them to the crime where none ex ists and to minimize the seriousness of the offense "by saying anyone else under similar conditions or circumstances might have done the same thing" (p. 97). In reviewing the recommendations made by Inbau, et al., Gudjons son (1994) states "this means that police officers are encouraged to make a false confession themselves in order to obtain a confession from suspects" (p. 239).

Another area problematic during interrogations is the reliance on meth ods for detecting deception (e.g., nonverbal behavior) that are offered in po lice training manuals. A suspect in police custody may be perceived by the interrogating officer as being deceptive when in fact this may not be true. In bau et al. (1986) recommend using the aforementioned techniques on sus pects who appear to be guilty based on the methods for detecting deception as described in their manual. However, research has shown that people--even those with special training--are poor at detecting deception (Ekman, 1992; Kassin & Fong, 1999; Shuy, 1998).

Negligence and overzealousness on the part of prosecutors (Bedau & Putnam, 1996; Frisman, 1995; Zimbardo, 1967) may also lead to false confes sions. Gardner (1995) recounts a case in which he was involved where the prosecution decided that a young woman was sexually abusing boys on the basis of a rumor and an anonymous tip. The alleged victims in the case were taken into police custody and told they would not be released until they "confessed" about the sexual abuses by this woman. Yant (1991) points out

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two major reasons why prosecutors abuse their power. First, American courts are structured as an adversarial system. The search for the truth is lost, and the focus becomes winning the case at any cost. In this effort, the prosecutor may "frequently cross the line into withholding or fabricating evidence, allowing perjured testimony, and making exaggerated attacks on the defendant" (p. 139). Second, in order to be re-elected or for other professional advancement reasons, many prosecutors feel they must maintain a high rate of convictions. In addition, prosecutors may encounter pressure for a political necessity to close a case (Greenspan, 1996; Ofshe, 1991). This would most likely occur in high profile cases where there is a public outcry for justice.

False Confessions: High Profile Cases

More than 60 years ago, over 200 people confessed to the kidnapping and murder of Charles Lindbergh's baby (Macdonald & Michaud, 1987; Rogge, 1959). Then in the late 1940s, more than 30 people falsely confessed to the murder and mutilation of Elizabeth Short, an aspiring Hollywood actress whose severed remains were found in a vacant Los Angles lot. The Short case received nationwide attention and became known as the "Black Dahlia" murder, due to descriptions of Ms. Short having always dressed in black. The Short case is still unresolved (Macdonald & Michaud, 1987; Nash, 1983; Rogge, 1959). Still another instance of a false confession is the story of SS leader Heinrich Himmler, who lost his pipe while visiting a concentration camp. A search followed, but upon returning to his car the pipe was discovered on his seat. The camp commandant protested that six prisoners have already confessed to stealing it (Macdonald & Michaud, 1987).

The above high profile cases are included to illustrate two general points to the reader. First, false confessions can and do occur, and they are not a new phenomenon. In fact, Munsterberg (1908) was the first psychologist to write on the subject nearly a century ago. In his classic book, *On the Witness Stand,* Munsterberg devotes an entire chapter to untrue confessions. Many of Munsterberg's observations on false confessions (e.g., "in some instances the confessing persons really believed themselves guilty" [p.146]; "pseudo-confessions may thus arise in men who are distinctly not ill" [p. 150]) are strikingly similar to what modern research has revealed on the subject. Second, false confessions occur in widely publicized types of cases in alarming numbers without any type of influence or pressure from the criminal justice system. When one takes this fact into account and further considers the influence of situational features (e.g., interpersonal pressure) during an interrogation, a better understanding of false confessions is fostered.

Types of False Confessions

What other reasons could explain why false confessions occur? Why do suspects who are innocent confess to crimes they did not commit? What is it about police interrogation that sometimes compels innocent people to in

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criminate themselves? A perusal of the anecdotal literature (Reik, 1959; Schafer, 1968; Zimbardo, 1967) has led Kassin and Wrightsman (1985) to distinguish among three psychologically distinct types of false confessions.

Voluntary False Confessions

A voluntary false confession is a self-incriminating statement that is purposefully offered in the absence of pressure by the police (Bedau & Putnam, 1996; Ofshe, 1992; Note, 1953; Kassin & Wrightsman, 1985; Wrightsman & Kassin, 1993). Canadian forensic law experts Rogers and Mitchell (1991) further noted that the accused who is giving a voluntary statement must have an operating mind--a mind that appreciates what is occurring and that appreciates the consequences of his or her action. Thus, the authors conclude, failing to understand or appreciate the consequences of expressing a statement only renders the statement inadmissible in court if the person does not possess an operating mind.

There are several possible reasons for why people give voluntary false confessions. A pathological need for fame and recognition (Radelet, Bedau, & Putnam, 1992) or as Note (1953) phrased a "morbid desire for notoriety" (p. 382) could account for the false confessions in cases which receive widespread public attention such as the Lindbergh kidnapping case and the "Black Dahlia" murder. Radelet et al. (1992) reported a case in which a man falsely confessed to a murder to impress his girlfriend. Gudjonsson (1999) conducted an extensive psychological evaluation of Henry Lee Lucas who is estimated to have confessed to over 600 murders. Gudjonsson concluded that Lucas "would say and do things for immediate gain, attention and reaction.....he was eager to please and impress people....the notoriety aspect of the confessions was appealing to him and fed into his psychopathology" (p. 423).

Frequently, false confessions are offered to protect a friend or relative, a fact often revealed in interviews with juvenile defenders (Gudjonsson, 1992; Gudjonsson & MacKeith, 1990). Huff, Rattner, & Sagarin, (1996) describe a scenario in which an innocent husband and wife are being held by police and the man falsely confesses to allow the wife to return home to tend to the children. Other possible motives for voluntary false confessions include an "unconscious need to expiate guilt over previous transgressions through self-punishment," (Kassin & Wrightsman, 1985, p. 77). Gudjonsson (1992) points out that a previous transgression can be eirther a real or an imagined act. Gudjonsson further states that the transgression does not necessarily have to be identifiable, "some individuals have a high level of generalized guilt, which is not related to a specific transgression, and this may influence a range of their behaviours [sic], including their need to volunteer a false confession" (p. 227). Finally, from my own experience, many individuals who have committed a crime that carries a large penalty will falsely confess to a lesser crime to avoid the more severe punishment associated with the original crime.

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Coerced-Compliant False Confessions

Coerced-compliant confessions occur when suspects confess, despite the knowledge of their innocence, due to extreme methods of police interrogations (Gudjonsson, 1991, 1992; Gudjonsson & MacKeith, 1990; Kassin, 1997; Kassin & Wrightsman, 1985; Wrightsman & Kassin, 1993). Numerous false confessions that were elicited through the use of torture, threats, and promises were presumed to be of this type, as in the Salem witchcraft confessions in the 17th century (Kassin & Wrightsman, 1985; Wrightsman & Kassin, 1993). The best known classic example of a coerced-compliant false confession is the case of *Brown v. Mississippi* (1936).

"Brainwashing," a technique commonly used on POWs falls under the category of coerced-compliant false confession. Almost forty years ago, during the Korean War, reports by the North Koreans stated that a number of captured American military men had confessed to a number of treasonable acts and expressions of disloyalty to the U. S. (Bem, 1966; Wrightsman & Kassin, 1993). Hunter (1960) examined the brainwashing methods used by the communists during the Korean War. The prisoners would attend communist indoctrination lectures, for a minimum of four hours, at least once per day. During these lectures, the prisoners would be forced to make a confession and express the communist point of view in his own words. The rationale behind forcing the prisoners to confess was to have it become second nature for them and become a part of their mentality. As Hunter (1960) points out:

> Each time a U.N. soldier stood up and used the words "I confess," his Red masters were confident that in the back of his mind a tiny trace at least of this intrinsic content of the world would filter down, even if only subconsciously. Each time he repeated it, they were certain a little more of this content was being rubbed onto his mentality. The communists actually heard him saying each time, in their double talk, "I submit," getting himself accustomed to the thought. (p. 238)

Similar confessions were made by some of the American POWs in the Vietnam War. During the first week of the Persian Gulf War in 1991, American TV viewers saw the grim and swollen faces of captured American airmen, and as reported by Fleming and Scott (1991), "each of the pilots identified himself and delivered a short speech deploring their government's involvement in Operation Desert Storm" (p. 127).

Coerced-Internalized False Confessions

The third type of false confession is coerced-internalized, that is, when suspects who are innocent, but anxious, fatigued, pressured, or confused, and then subjected to highly suggestive methods of police interrogation, actually come to believe that they committed the crime (Kassin, 1997; Kassin & Kiechel, 1996; Kassin & Sukel, 1997; Kassin & Wrightsman, 1985; Wrightsman & Kassin, 1993). What is frightening about this type of false confession is that the innocent suspects' memory of their own actions may

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be altered, making its "original contents potentially irretrievable." (p. 226, Kassin, 1997; p. 78, Kassin & Wrightsman, 1985).

There are remarkable cases involving coerced-internalized false confes sions. Kassin (1997) asserts that they all have two factors in common, namely: (a) a suspect who is vulnerable--i.e., one whose memory is malle able by virtue of his/her youth, interpersonal trust, naiveté, suggestibility, lack of intelligence, stress, fatigue, alcohol, or drug use, and (b) the presen tation of false evidence such as a rigged polygraph or other forensic tests (e.g., bloodstains, semen, hair, fingerprints), statements supposedly made by an accomplice, or a staged eyewitness identification as a way to convince the beleaguered suspect that he or she he is guilty. (p. 227)

Until recently, there was no empirical evidence for the concept of co erced-internalized false confessions. However, eyewitness memory re searchers have found that misleading post-event information can alter actual or reported memories of observed events (Cutler & Penrod, 1995; Loftus, 1979; Loftus & Ketcham, 1994). Recent studies suggest that it is even pos sible to implant false "recollections" of isolated childhood experiences, such as being lost in a shopping mall, that supposedly had been forgotten or bur ied in the unconscious, but in reality never happened (Loftus & Ketcham, 1994).

Various theories have been developed to respond to the question: What is it about police interrogation that cause some innocent people to incrimi nate themselves? From a psychological viewpoint (e. g., Kassin, 1997; Kassin & Wrightsman, 1985; Wrightsman & Kassin, 1993), coerced-compliant false confessions are explained by the innocent suspect's wish to escape an aver sive situation and ensure a pleasant consequence. But what about the more baffling examples of internalized false confessions?

To account for the phenomenon of internalized false confessions, some observers (e. g., Ofshe, 1992; Kassin & Wrightsman, 1985; Wrightsman & Kassin, 1993) have compared the interrogation process to hypnosis. Foster, referring to the "station house syndrome," asserted that police questioning "can produce a trance-like state of heightened suggestibility in the suspect" so that "truth and falsehood become hopelessly confused in the suspect's mind" (1969, pp. 690-691). A. A. Liebault was a physician in the 1860s who believed that hypnosis was based on the implantation of a fixed idea in the mind of the subject. The subject relinquished his or her freedom of choice and carried out any suggestion that had been implanted in their mind (Laur ence & Perry, 1988). In 1970, Weinstein, Abrams, and Gibbons discovered that when a false sense of guilt is introduced into the minds of hypnotized individuals, they fair less in a polygraph lie detector test.

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Interrogative Suggestibility

Gudjonsson and Clark (1986) proposed the concept of interrogative suggestibility to account for the individual differences in responses to police questioning. Gudjonsson defines interrogative suggestibility as "the extent to which within a closed social interaction, people come to accept messages communicated during formal questioning, as the result of which their subsequent behavioral response is affected" (1991, p. 280). Gudjonsson (1991) reported five interrelated components that are part of the concept of interrogative suggestibility: (a) a closed social interaction between the interrogator and the interviewee, (b) a questioning procedure that involves two or more participants, (c) a suggestive stimulus, [e.g., a specific influential message (Schumaker, 1991) or a hint, cue, or idea (Gudjonsson, 1992)], (d) acceptance of the suggestive stimulus, and (e) a behavioral response to indicate whether or not the suggestion is accepted (p. 280). Gudjonsson (1991) further explains that interrogative suggestibility differs from other types of suggestibility in four ways: (a) the above-mentioned closed nature of the social interaction, (b) the questions asked deal mainly with past experiences and recollections, (c) the situation has a component of uncertainty, and (d) the situation is stressful with important consequences for the person being interviewed. In this situation, the interrogator can manipulate three aspects--uncertainty, interpersonal trust, and expectation--to alter the person's susceptibility to suggestions.

But characteristics of the person being interviewed also affect the level of his or her suggestibility, (e.g., people who are suspicious are less suggestible than those who are trusting). Those with low intelligence and poor memories are generally more suggestible; low self-esteem, lack of assertiveness, and anxiety also affect suggestibility (Gudjonsson, 1991, 1999). Moreover, Gudjonsson observes those who were most suggestible as having:

> . . . failed to be able to evaluate each question critically and give answers that to them seemed plausible and consistent with the external cues provided. Nonsuggestible subjects, on the other hand, were able to adopt a critical analysis of the situation which facilitated the accuracy of their answers. (1991, p. 285)

An example can be found in the case of Delbert Ward, an introverted, easygoing but frail 59-year-old farmer with an IQ of 69 and functioning only in the "educably mentally retarded" range. Following long hours of intense questioning and surrounded by five or six 250-pound state troopers, Ward signed a false confession of murdering his own brother. Forensic pathologist Cyril Wecht (1994), stated in his book, *Cause of Death,* that according to Dr. Blumetti, the clinical psychologist who interviewed Ward, "Delbert would likely have been so nervous and confused at the time of his interrogation that he would have agreed with anything." [He added that his ability to reason was impaired and that his major focus would have been] "to get out of

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that setting. . . . His focus would not be on the questions he was being asked, but on getting out of that unfamiliar, threatening environment" (pp. 255-256).

Internalized false confessions, from another viewpoint, could result from a process of self-perception. Bem (1966) probed the idea that a false confession could alter the recollection of a person's past behavior if the confession is given in the presence of cues previously associated with telling the truth (e.g., reassurance that one need not admit to wrongdoing). The result of his experiment led Bem to conclude that under conditions normally associated with telling the truth, subjects came to believe the lies they had been induced to tell. Bem (1967) further noted that "saying becomes believing only when we feel the presence of truth, and certainly only when a minimum of inducement and the mildest and most subtle forms of coercion are used" (pp. 23-24). Bem's self-perception theory partially explains the internalized false confession phenomenon (Reifman, 1998). Closely related to Bem's theory is an interrogation tactic described by Driver (1968) of having the suspect repeat the story over and over, for "if duped into playing the part of the criminal in an imaginary sociodrama, the suspect may come to believe that he was the central actor in the crime" (p.53). Another factor to consider in regard to internalized false confessions is the two-factor theory of emotions proposed by Schachter and Singer (1962). According to this theory, the experience of emotion depends on an interaction of two factors: (1) physiological arousal and (2) cognitive processes. As a result of this interaction, the individual is thought to experience an emotional state. Suggestible individuals during a police interrogation may cognitively label physiological response as guilt and would conclude that they are feeling guilty therefore they must have had some involvement in the crime. The result may be an internalized false confession.

Other Psychological Factors that Influence Innocent Suspects to Confess Falsely

Eysenck (1964) concurs with Gudjonsson that innocent suspects that have certain personalities and characteristics are also more prone to suggestibility, and are thus more likely to give false statements and confess to crimes that they did not commit. According to Eysenck, introverts are capable of being conditioned more easily than extroverts. Since most criminals are extroverts, the methods of interrogation that are designed to effectively deal with the typical extrovert criminal may have an overwhelming impact on a suspect who is an easily-conditionable introvert. Therefore, in addition to children, the mentally retarded (Perske, 1994) and the feeble-minded, perhaps individuals who are exceptionally introverted, as in the case of Delbert Ward (Wecht, 1994), are also at risk. Perhaps a certain amount of stress applied to a normal person may get the truth out of him or her; but if a lot of stress is applied to the psychologically inadequate, the result could likely be a false confession (Brandon & Davies, 1973, Gudjonsson, 1992). The aforementioned psychological theories suggest some of the factors that may influ

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ence innocent suspects to give false confessions. What will be explored next are the types of psychological and social influence police interrogators use to get suspects to confess.

For law enforcement officials (Kassin, 1997), the purpose of interrogation is twofold: to obtain a full or partial confession and to elicit information on other evidence that is relevant to a case. Observational studies (e. g., Driver, 1968; Kassin, 1997; Kassin & Wrightsman, 1985; Wrightsman & Kassin, 1993; Zimbardo, 1967) have shown that the use of physical force has given way to more psychologically oriented methods, such as feigned sympathy and friendship, appeals to God and religion, the use of informants, the presentation of false evidence, and other forms of trickery and deception (Leo, 1992). After spending a year following homicide detectives in Baltimore, Simon (1991) described the police interrogator as:

> . . . a salesman, a huckster as thieving and silver-tongued as any man who ever moved used cars or aluminum siding, more so, in fact, when you consider that he's selling long prison terms to customers who have no genuine need for the product. (p. 213)

Many observers contend that deceptive and deceitful practices being used by police during the interrogation process may result in false confessions (e. g., Bedau & Putnam, 1994; Brandon & Davies, 1973; Driver, 1968; Frisman, 1995; Greenspan, 1996; Kassin, 1997; Kassin & Sukel, 1997; Macdonald, 1969; Macdonald & Michaud, 1987; McCann, 1998; Ofshe, 1991; Rogers & Mitchell, 1991; Schafer, 1968; Underwager & Wakefield, 1992; Wecht, 1994; Wood, 1995; Zimbardo, 1967). The polygraph or "lie-detector" and truth serum tests are some of the practices often manipulated by the police (Inbau, Reid, & Buckley, 1986; Macdonald, 1969; Macdonald & Michaud, 1987). Underwager and Wakefield (1992) have seen several cases through their analysis of videotapes, audiotapes, and documents of actual police interrogations, some in which the accused were falsely told that they had failed the lie detector test and should therefore confess. However, in these cases the lie detector test was not failed, but proved to be inconclusive.

Most people have no idea "how incompletely and inaccurately (they) understand the way lie detectors really work. . . . (they) share the popular misconception that polygraphs don't make mistakes..." (Radelet, Bedau, & Putnam, 1992, p. 221). However, contrary to popular belief, polygraphs *do* make mistakes (Bedau & Putnam, 1996; Radelet et al., 1992; Underwager & Wakefield, 1992). The person who conducts the test and interprets the data may affect the outcome of the polygraph. It would be particularly troubling if the polygraph examiner began the examination with a preconception that the suspect was likely guilty. An egregious misuse of the polygraph would be to use it only as an interrogative wedge to move a person already assumed to be guilty toward a confession.

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The term "truth serum" was coined to describe the use of scopolamine, a type of drug used as a means of obtaining confessions from criminals and of exonerating the innocent. The term has since been applied to any drug which is employed to obtain confessions. A drugged person is highly suggestible, and this type of condition may lead to false or misleading answers especially when the questions are phrased improperly (Macdonald, 1969). Inbau, Reid, and Buckley (1986) opined that such tests are often effective on persons who, if properly interrogated, would have been truthful anyway. The person who is determined to lie will usually be able to continue the deception even under the influence of the drug. On the other hand, the person who is likely to confess will probably do so as the result of skillful police interrogation, and it will not be necessary to use drugs.

The length of the interrogation may also have an impact on its outcome. In several of the cases noted, such as *Chambers v. Florida* (1940), the suspect was subjected to five days of prolonged questioning before a confession was obtained. This technique is commonly known as the "wear down" process and involves detaining an individual for a lengthy period of time whereas the focus of the individual becomes on short-term gratification (i.e., removing oneself from the present situation) while failing to consider the long-term consequences (i.e., a possible sentence). While referring to such a process concerning the Salem witch trials, Munsterberg (1908) commented: "In tedious examinations the prisoners were urged to confess through many hours till the accused were wearied out by being forced to stand so long or by want of sleep" (p. 148). Brandon & Davies (1973) observe that "almost anybody could be worn down by such a process if it goes on long enough, and is tough enough" (p. 52). Gudjonsson and Sigurdsson (1994) found that the most common reason inmates in an Icelandic prison made a false confession was to escape police pressure and to get out of police custody. It my own belief that an innocent suspect could be made to admit almost anything under the pressure of continuous questioning and suggestion. The individual would experience a feeling of incompetence that would increase feelings of helplessness and lack of control over the situation and then simply "submit."

The Court's Opinion on Police Interrogation

The Supreme Court of the United States, upon making its decision in the landmark case *Miranda v. Arizona* in 1966, quoted from the most prominent textbook, of that time, for training police officers, Inbau and Reid's (1962) *Criminal Interrogation and Confessions,* to show that police used deception and psychologically coercive methods in questioning suspects (Underwager & Wakefield, 1992). The Court concluded that police questioning is oriented psychologically rather than physically, but that the rate of duress inherent in the situation was not diminished; recent observers concur (e. g., Kassin, 1997; Kassin & Wrightsman, 1985; Underwager & Wakefield, 1992). In *Miranda v. Arizona* (1966), the Supreme Court held that a confession, ob

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tained from a suspect in custody during police interrogation, was admissible only if it was made voluntarily, not coercively, and only if the police had taken the appropriate steps to ensure protection of the rights of the accused under the self-incrimination clause of the Fifth Amendment (Driver, 1968; Kassin, 1997; Kassin & Wrightsman, 1985; Underwager & Wakefield, 1992), That is, the police must advise suspects in custody of their constitutional rights to silence and to counsel. Law enforcement advocates immediately protested that this decision would handcuff the police in their efforts to elicit confessions (Kassin, 1997). Legal scholars soon concluded that the Supreme Court's ruling was not having this effect. In fact, research suggested that many juvenile suspects did not fully understand the rights they were given (Grisso, 1981, 1998).

Today, the Miranda issue is still in dispute. Critics of the Miranda warnings maintain that the confession and conviction rates have dropped significantly as a direct result of the warning and waiver requirements, thus triggering the release of dangerous criminals (Cassell, 1996). Proponents of the Miranda warnings, on the other hand, argue that the actual declines are insubstantial (Schulhofer, 1996), that four out of five suspects waive their rights and submit to questioning, and that the Miranda decision has had a civilizing effect on police interrogation practices and has increased public awareness of constitutional rights (Leo, 1996).

Possible Consequences During Police Interrogation

Being interrogated by the police is a highly stressful experience. This stress can worsen when the suspect is isolated. Gudjonsson and MacKeith (1990) agree that isolation and confinement can cause a wide range of behavioral and physiological disturbances including loss of contact with reality. Furthermore, they note that factors encouraging a suspect to make a genuine confession may be similar to those influencing a person to make a false confession. They state that "non-psychotic individuals ruminating guiltily about such things as sexual deviation may also have an exceptionally low threshold to confession to things that they have not actually done." (cited in Underwager & Wakefield, 1992). The false confessor may be aware that she or he is not telling the truth or his or her perceptions may be distorted or she or he might even be deluded for a brief period of time. A false confession in all of these situations is an interplay between the person's mental state, basic personality, intelligence, and all of the circumstances of the interrogation (Gudjonsson & MacKeith, 1990).

Demand Characteristics of Police Interrogation

In order to increase the likelihood of a confession from a suspect by police, the following conditions must be met. During questioning, an environment that minimizes sensory stimulation, maximally exposes the suspect's vulnerability, and provides for complete control and domination by the interrogator must be created (Zimbardo, 1967). Privacy, being totally alone

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with suspects, is therefore highly imperative, for it is the primary psychological factor conducive to successful interrogations (Inbau & Reid, 1967; Tousignant, 1991). Several authors (e. g., Inbau & Reid, 1967; Inbau et al., 1986; Macdonald, 1969; Tousignant, 1991; Zimbardo, 1967) concur that the location must have surroundings that are unfamiliar to the suspect. The interviewing room must be free from noise, must have no windows, and must be bare except for a table and a couple of chairs: one for the suspect and one for the interrogator. The interrogator must try to establish a superficial friendship with the suspect as well as exhibit unexpected kindness. The former must also feign the seriousness of the crime by excusing the crime (minimization of seriousness). Other strategies include employing the sympathetic approach, trickery, and deceit (Inbau & Reid, 1967; Inbau et al., 1986; Macdonald, 1969; Macdonald & Michaud, 1987).

The above-mentioned are only part of the demand characteristics of police questioning and are constituents of the 16 strategies for interrogation proposed by Inbau, Reid, and Buckley's manual (1987). According to Underwager and Wakefield (1992), the U. S. Supreme Court noted that these 16 strategies show three major recurring themes in the manual: 1. The first is to reattribute the implications of the situation by shifting the blame or minimizing the seriousness. 2. Alternatively, the questioning may aim at frightening the individual by exaggerating the evidence available, the consequences to the individual, or stating firmly that the interrogator knows the person is guilty. 3. The third theme is the emotional appeal to the person being questioned by showing sympathy, flattery, respect, and appeal to the best interest of the suspect. These are some of the practices the court found inherently coercive. (p. 166)

The police methods designed to obtain confessions can potentially undermine the concept of a voluntary confession. Based on his review of training manuals, Zimbardo (1967) believes that the interrogation techniques of the police are sometimes more highly developed, more psychologically sophisticated, and more effective than those that were used by the Chinese Communists in Korea.

Summary

There are a significant number of wrongful convictions in the United States. A 1987 study by Bedau and Radelet identified that the primary cause for the conviction of 49 of the 350 cases of miscarriages of justice in the U. S. was a false confession obtained by coercive police questioning. How often false confessions result in wrongful convictions is unknown, although some observers (e. g., Kassin & Wrightsman, 1985; Wrightsman & Kassin, 1993; Wrightsman et al., 1994) attest that enough cases have been documented to suggest that a concern over such a risk is justified. A review of the literature indicated that there are a limited number of research studies and little

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empirical data available for extensive study in the psychology of false confes sions.

There are psychological and social factors that influence innocent sus pects to give self-incriminating false statements such as suggestibility. There are other variables as well. Deception and deceit, together with other questionable and inappropriate police interrogation and investigative proce dures seem to be common and continue to be employed during the interroga tion process. An indeterminate number of false confessions may be attrib uted to the inherently coercive nature of police interrogation during which deceptive and deceitful practices may be used and approved by the judicial system.

Conclusions

First, in view of the current research findings on false confessions, one must take a closer look at the formidable and detrimental impact of ques tioning techniques employed by police interrogators and investigators. Sev eral studies (Baldwin, 1993; Fisher, Geiselman, & Raymond, 1987; Moston, Stephenson, & Williamson, 1992) have shown that overall, police investiga tors possess poor interviewing skills. Some of the more questionable inter viewing techniques noted in the studies included interrupting the suspect, asking questions in a rapid manner (Baldwin, 1993; Fisher, Geiselman, & Raymond, 1987), possessing a limited degree of flexibility during the inter view (Moston, Stephenson, & Williamson, 1992), inappropriate sequencing of questions, negative phrasing of questions, nonneutral wording of questions, inappropriate language, and judgmental comments (Fisher, Geiselman, & Raymond, 1987). In light of the results of these studies and the fact that po lice spend as much as 85% of their on duty time talking to people (Miline & Bull, 1999), it seems apparent that police investigators should receive spe cial training in appropriate interviewing skills and be instructed of the dan gers of not using such skills. During training, special attention should also be given to dealing with individuals with special needs such as the mentally handicapped. In the UK, this issue has been addressed. Juveniles, the men tally ill, and the mentally handicapped are identified as "at risk," and during interrogation, an appropriate adult (e.g., parent, social worker) must be pre sent to assist with communication and safeguard the rights of the individual (McKenzie, 1994). Effective communication practices by investigators will lead to accuracy (Shuy, 1998) and accountability in the criminal justice sys tem and hopefully reduce the number of erroneous convictions.

Second, the judicial system needs to be more cognizant of the inappropri ate approaches of eliciting confessions from suspects in custody. Interroga tions should be centered around eliciting the truth rather than attempting to secure a confession. When questioning a potential suspect, the investigator should thus assume a disinterested role rather than an adversarial one. Re search has shown that many of the tactics used to persuade a suspect who

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is reluctant to volunteer information described in popular interrogation manuals such as Inbau, et al. (1986), are quite unnecessary and fail to per suade suspects to alter their initial response given to police investigators (Baldwin, 1993; Moston, Stephenson, & Williamson, 1992). In reviewing 600 police interrogations in the UK, Baldwin (1993) found that only three sus pects were persuaded to change their initial statements during an interview due to the persuasive skills of the interrogator. Baldwin calls for such inter rogation techniques to be outlawed in order for the concern over miscarriages of justice to be minimized.

Related to this issue is the length of the interrogation process which also seems to have an impact on the possibility of a false confession. The longer the interrogation process, the greater the likelihood of an untrust worthy confession (Leo & Ofshe, 1998; Munsterberg, 1908; White, 1998). In many of the cases presented in the Bedau & Radelet (1987) study, the inter rogations of suspects lasted for several hours and in some cases several days. Prolonged interrogations were also used in several of the landmark cases presented earlier (i.e., *Chambers v. Florida*). It is important to note that in Baldwin's (1993) study, almost 75% of the interviews were concluded within 30 minutes, which indicates that interrogations can be completed in a reasonable amount of time. In the UK, there are guidelines which limit the length of interrogations (36 hours; 96 with court approval) and the time of day in which the interrogations may take place--they may not take place when the individual would normally be sleeping (McKenzie, 1994). Similar guidelines in the United States could perhaps curb the possibility of a co erced confession and save on police resources.

Third, in order to eliminate bias and to ensure the accuracy and authen ticity of confessions, it is imperative that statements issued be corroborated by evidence. In several of the cases reviewed (e.g., *Brown v. Mississippi*) and in a substantial number of cases from studies cited (e.g., Bedau & Radelet, 1987), many of the defendants were tried, convicted, and sentenced on the basis of a confession alone; there was no physical evidence linking them to the crime in question. Gudjonsson (1992) points out that in Scotland an in dividual can not be convicted solely on the basis of a confession. Such a pro cedural safeguard in the United States would relieve any doubts about the authenticity of a suspect's confession.

Fourth, in order to ensure the validity and veracity of the obtained con fessions, I propose a videotaping or audiotaping of *all* interrogations. Oppo nents of videotaping argue the practice would discourage confessions from suspects and be quite costly (Higgins, 1998). However, an exploratory study on videotaping interrogations and confessions by Geller (1993) found that 63.1% of the police agencies surveyed reported no change in suspects' will ingness to talk, and 60% reported that more incriminating information was given by suspects while being videotaped. The study also found that since the adoption of videotaping, claims of police misconduct were reduced, and 97% of police agencies reported videotaping to be useful. Based on the re

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search, it would seem that a mandatory videotaping requirement would serve a dual purpose of protecting police agencies from claims of misconduct and safeguarding the rights of suspects. The costs of implementing videotaping would be offset by reducing officer stress and burnout (Geller, 1993), not to mention the costs associated with exonerating an individual who has been incarcerated. As of this writing, in the United States, only Alaska and Min nesota require the videotaping of interrogations (Higgins, 1998), while in England and Wales the recording of suspect interviews is mandatory (McKenzie, 1994).

Finally, additional research on the subject of false confessions is re quired. To date, only one laboratory experiment has been conducted on the subject. Kassin and Kiechel (1996) hypothesized that:

> the presentation of false evidence can lead individuals who are vul nerable (i.e., in a heightened state of uncertainty) to confess to an act they did not commit and, more important, to internalize the confes sion and perhaps confabulate details in memory consistent with that new belief. (p. 126)

To test this hypothesis, Kassin and Kiechel (1996) asked 79 students to participate in a reaction time experiment. After being warned not to touch the "ALT" key because the computer would crash, a confederate read letters to the participants in two different speeds to manipulate the participant's vulnerability. After 60 seconds, the computer was purposefully crashed by the experimenters. None of the participants was responsible for causing the computer to crash; however, each was blamed for doing so.

Half of the participants were told that the confederate had seen them hit the "ALT" key. Participants were then asked to sign a handwritten confes sion that they had hit the "ALT" key causing the computer to crash. Overall, 69% of the subjects signed the confession, 28% internalized their guilt (be lieving they had hit the wrong button), and 9% confabulated details to fit with their false belief that they had caused the computer to crash. The most vul nerable group was the fast typing/false evidence (having been "seen" hit the key); 100% of this group signed the confession. This study supported the no tion that when presented with false evidence people can be induced to inter nalize guilt for an event with which they had no involvement. Kassin and Kiechel (1996) also recommend that additional research is needed to exam ine other methods commonly used in police interrogation manuals (e.g., minimization) and other risk factors (i.e., sleep deprivation, etc.) that could possibly lead to an unreliable confession. It is also hoped that in the future, with the increased use of videotaping interrogations, further research can be conducted to clarify some of the gray areas regarding false confessions. This research could perhaps facilitate appropriate legislation in the regulation of questionable interrogation tactics.

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References

Anderson, S. M., Lepper, M. R., & Ross, L. (1980). Perseverance of social theories: The role of explanation in the persistence of discredited information. *Journal of Personality and Social Psychology, 39,* 1037-1049.

Arizona v. Fulminante, 499 U.S. 279 (1991).

Baldwin, J. (1993). Police interview techniques: Establishing truth or proof? *The British Journal of Criminology, 33,* 325-352.

Bedau, H. A., & Putnam, C. (1996). False confessions and other follies. In D. S. Connery (Ed.), *Convicting the innocent* (pp. 69-83). Cambridge, MA: Brookline Books.

Bedau, H. A., & Radelet, M. L. (1987). Miscarriages of justice in potentially capital cases. *Standford Law Review, 40,* 21-179.

Bem, D. J. (1966) Inducing belief in false confessions, *Journal of Personality and social psychology, 3,* 707-710.

Bem, D. J. (1967). When saying is believing. *Psychology Today, 1*(2), 21-25.

Brandon, R., & Davies, C. (1973). *Wrongful imprisonment.* London, England: Allen and Unwin Brothers Ltd.

Brown v. Mississippi, 297 U.S. 278 (1936).

Cassell, P. G. (1996). All benefits, no costs: The grand illusion of Miranda's defenders. *Northwestern University Law Review, 90, 1084-1124.*

Chambers v. Florida, 309 U.S. 277 (1940).

Connery, D. S. (Ed.). (1996). *Convicting the innocent.* Cambridge, MA: Brookline Books.

Culombe v. Connecticut, 367 U.S. 568 (1961).

Cutler, B. L., & Penrod, S. D. (1995). *Mistaken identification: The eyewitness, psychology, and the law.* New York: Cambridge University Press.

Driver, E. D. (1968). Confessions and the social psychology of coercion, *Harvard Law Review, 82* (42), 42-61.

Ekman, P. (1992). *Telling lies.* New York: W. W. Norton.

Eysenck, H. J. (1964). *Crime and personality.* London: Routledge.

Federal Bureau of Investigation, (1997). *Uniform crime reports: Crime in the United States. Washington,* D. C.: U. S. Department of Justice.

Fisher, R. P., Geiselman, R. E., & Raymond, D. S. (1987). Critical analysis of police interview techniques. *Journal of Police Science and Administration, 15,* 177-185.

Fleming, J. H., & Scott, B. A. (1991). The costs of confession: The Persian Gulf War POW tapes in historical and theoretical perspective. *Contemporary Social Psychology, 15,* 127-138.

Foster, H. H. (1969). Confessions and the station house syndrome. *Depaul Law Review, 18,* 683-701.

Frisman, P. (1995). Easy confessions make tough law. In D. S. Connery (Ed.), *Convicting the innocent.* Cambridge, MA: Brookline Books.

Gardner, R. A. (1995). *Testifying in court: A guide for mental health professionals.* Cresskill, NJ: Creative Therapeutics.

Geller, W. A. (1993). *Videotaping interrogations and confessions. National Institute of Justice: Research in Brief.* Washington, DC: U.S. Department of Justice.

Gilbert, D. T., & Malone, P. S. (1995). The correspondence bias. *Psychological Bulletin, 117,* 21-38.

The Journal of Credibility Assessment and Witness Psychology
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Greenspan, S. (1996). There is more to intelligence than IQ. In D. S. Connery (Ed.), *Convicting the innocent.* Cambridge, MA: Brookline Books.

Grisso, T. (1981). *Juveniles' waiver of rights: Legal and psychological competence.* New York: Plenum.

Grisso, T. (1998). *Forensic evaluation of juveniles.* Sarasota, FL: Professional Resource Press.

Gudjonsson, G. H. (1991). The application of interrogative suggestibility to police interviewing. In J. F. Schumaker (Ed.), *Human suggestibility: Advances in theory, research, and application* (pp. 279-288). New York: Routledge.

Gudjonsson, G. H. (1992). *The psychology of interrogations, confessions, and testimony.* New York: Wiley.

Gudjonsson, G. H. (1994). Investigative interviewing: Recent developments and some fundamental issues. *International Review of Psychiatry, 6,* 237-245.

Gudjonsson, G. H. (1999). The making of a serial false confessor: The confessions of Henry Lee Lucas. *The Journal of Forensic Psychiatry, 10,* 416-426.

Gudjonsson, G. H., & Clark, N (1986). Suggestibility in police interrogation: A social psychological model. *Social Behaviour, 1,* 83-104.

Gudjonsson, G. H., & MacKeith, J. A. C. (1988). Retracted confessions: Legal, psychological, and psychiatric aspects. *Medicine, Science, and the Law, 28,* 187-194.

Gudjonsson, G. H., & MacKeith, J. A. C. (1990). A proven case of false confession: Psychological aspects of the coerced-compliant type. *Medicine, Science, and the Law, 30,* 329-335.

Gudjonsson, G. H., & Sigurdsson, J. F. (1994). How frequently do false confessions occur? An empirical study among inmates. *Psychology, Crime and Law, 1,* 21-26.

Haynes v. Washington, 373 U.S. 503 (1963).

Higgins, M. (1998). Irrefutable evidence. *ABA Journal, 84,* 18-20.

Huff, C. R., Rattner, A., & Sagarin, E. (1986). Guilty until proved innocent: Wrongful conviction and public policy. *Crime & Delinquency, 32,* 518-544.

Huff, C. R., Rattner, A., & Sagarin, E. (1996). *Convicted but innocent: Wrongful conviction and public policy.* Thousand Oaks, CA: Sage.

Hunter, E. (1960). *Brainwashing: From Pavlov to Powers.* Linden, NJ: Bookmailer.

Inbau, F. E., & Reid, J. E. (1962). *Criminal interrogation and confessions.* (1st ed.). Baltimore: Williams & Wilkins.

Inbau, F. E., & Reid, J. E. (1967). *Criminal interrogation and confessions.* (2nd ed.). Baltimore: Williams & Wilkins.

Inbau, F. E., Reid, J. E., & Buckley, J. P. (1986). *Criminal interrogation and confessions.* (3rd ed.). Baltimore: Williams & Wilkins.

Kassin, S. M. (1997). The psychology of confession evidence. *American Psychologist, 52,* 221-233.

Kassin, S. M. (1998). More on the psychology of false confessions. *American Psychologist, 53,* 320-321.

Kassin, S. M., & Fong, C. T. (1999). "I'm innocent!": Effects of training on judgments of truth and deception in the interrogation room. *Law and Human Behavior, 23,* 499-516.

Kassin, S. M., & Kiechel, K. L. (1996). The social psychology of false confessions: Compliance, internalization, and confabulation. *Psychological Science, 7,* 125-128.

Kassin, S. M., & Sukel, H. (1997). Coerced confessions and the jury: An experimental test of the "harmless error" rule. *Law and Human Behavior, 21*(1), 27-46.

Kassin, S. M., Williams, L. N., & Saunders, C. L. (1990). Dirty tricks of cross-examination: The influence of conjectural evidence on the jury. *Law and Human Behavior, 14,* 373-384.

Kassin, S. M., & Wrightsman, L. S. (1985). Confession evidence. In S. M. Kassin and L. S. Wrightsman (Eds.), *The psychology of evidence and trial procedure* (pp. 67-94). Beverly Hills: Sage.

Laurence, J. R., & Perry, C. (1988). *Hypnosis will and memory.* New York: Guilford Press.

Leo, R. A. (1996). Inside the interrogation room. *The Journal of Criminal Law and Criminology, 86,* 621-692.

Leo, R. A. (1998). Miranda and the problem of false confessions. In R. A. Leo & G. C. Thomas III (Eds.), *The Miranda debate: Law, justice, and policing* (pp. 271-282). Boston: Northeastern University Press.

Leo, R. A., & Ofshe, R. J. (1998) The consequences of false confessions: Deprivations of liberty and miscarriages of justice in the age of psychological interrogation. *Journal of Criminal Law and Criminology, 88,* 429-497.

Lloyd-Bostock, S. (1989). *Law in practice.* Chicago: Lyceum.

Loftus, E. F. (1979). *Eyewitness testimony.* Cambridge, MA: Harvard University Press.

Loftus, E. F., & Ketcham, K. (1994). *The myth of repressed memory.* New York: St. Martin's.

Macdonald, J. M. (1969). *Psychiatry and the Criminal.* (2nd ed.). Springfield, IL: Charles C. Thomas.

Macdonald, J. M., & Michaud, D. L. (1987). *The confession: Interrogation and criminal profiles for police officers.* Denver, CO: Apache.

McCann, J. T. (1998). Broadening the typology of false confessions. *American Psychologist, 53,* 319-320.

McCormick, C. T. (1972). *Handbook of the law of evidence* (2nd ed.). St. Paul, MN: West.

McCormick, C. T. (1992). *Handbook of the law of evidence.* (4th ed.). St. Paul, MN: West.

McKenzie, I. K. (1994). Regulating custodial interviews: A comparative study. *International Journal of the Sociology of Law, 22,* 239-259.

Milne, R., & Bull, R. (1999). *Investigative interviewing: Psychology and practice.* New York: Wiley.

Miranda v. Arizona, 384 U.S. 436 (1966).

Moston, S., Stephenson, G. M., & Williamson, T. M. (1992). The effects of case characteristics on suspect behaviour during police questioning. *British Journal of Criminology, 32,* 23-40.

Munsterberg, H. (1908) *On the witness stand.* New York: Doubleday.

Nash, J. R. (1983). *Open files: A narrative encyclopedia of the world's greatest unsolved crimes.* New York: McGraw-Hill.

Note (1953). Voluntary false confessions: A neglected area in criminal administration. *Indiana Law Journal, 28,* 374-392.

Ofshe, R. (1991). I'm guilty if you say so. In D. S. Connery (Ed.), *Convicting the innocent* (pp. 95-108). Cambridge, MA: Brookline Books.

Perske, R. (1994). Thoughts on the police interrogation of individuals with mental retardation. *Mental Retardation, 32,* 377-380.

Radelet, M. L., Bedau, H. A., & Putnam, C. E. (1992). *In spite of innocence.* Boston: Northeastern University.

Reifman, A. (1998). Social psychology of false confessions: Bem's early contribution. *American Psychologist, 53,* 320.

Reik, T. (1959). *The compulsion to confess.* New York: Farrar, Straus, and Cudahy.

Rogers, R., & Mitchell, C.N. (1991). *Mental health experts and the criminal courts.* Ontario, Canada: Thomson Professional.

Rogge, O. J. (1959). *Why men confess.* New York: Da Capo Press.

Ross, L. D. (1977). The intuitive psychologist and his shortcomings: Distortions in the attribution process. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 10). New York: Academic Press.

Schachter, S., & Singer, J. E. (1962). Cognitive, social and physiological determinants of emotional state. *Psychological Review, 69,* 379-399.

Schafer III, W. J. (1968). *Confessions and statements.* Springfield, IL: Charles C. Thomas.

Schulhofer, S. J. (1996). Miranda's practical effect: Substantial benefits and vanishingly small social costs. *Northwestern University Law Review, 90,* 500-564.

Schumaker, J. F. (Ed.). (1991). *Human suggestibility: Advances in theory, research, and application* . New York: Routledge.

Shuy, R. W. (1998). *The language of confession, interrogation, and deception.* Thousand Oaks, CA: Sage.

Simon, D. (1991). *Homicide: A year on the killing streets.* New York: Ivy Books.

Tousignant, D. D. (1991, March). Why suspects confess. *FBI Law Enforcement Bulletin,* 14-18.

Underwager, R., & Wakefield, H. (1992). False confessions and police deception. *American Journal of Forensic Psychology, 10*(3), 163-174.

Wecht, C. (1994). *Cause of Death.* New York: Onyx Books.

Weinstein, E., Abrams, S., & Gibbons, D. (1970). The validity of the polygraph with hypnotically induced repression and guilt. *American Journal of Psychiatry, 126,* 1159-1162

White, W. S. (1998). What is an involuntary confession now? *Rutgers Law Review, 50,* 2001-2057

Wood, A. (1995). Lying accepted investigative tool. In D. S. Connery (Ed.), *Convicting the innocent* (pp. 39-45). Cambridge, MA: Brookline Books.

Wrightsman, L. S. (1991). *Psychology and the legal system* (2nd ed.). Belmont, CA: Brooks/Cole.

Wrightsman, L. S., & Kassin, S. M. (1993). *Confessions in the courtroom.* Newbury Park, CA: Sage.

Wrightsman, L. S., Nietzel, M. T., & Fortune, W. H. (1994). *Psychology and the legal system.* (3rd ed.). Pacific Grove, CA: Brooks/Cole.

Yant, M. (1991). *Presumed guilty: When innocent people are wrongly convicted.* Buffalo, NY: Prometheus.

Zimbardo, P. G. (1967). The psychology of police confessions. *Psychology Today, 1*(2), 17-20, 25-27.

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TAB 16

Melissa Lucio – Age 1-6

Father of Melissa absent from the home and father of Sonia and Diane. Mother working. Sonia's Father died before Diane born..

Mother absent most of time. Mother working; children with multiple caretakers.

Functional relationship

No relationship

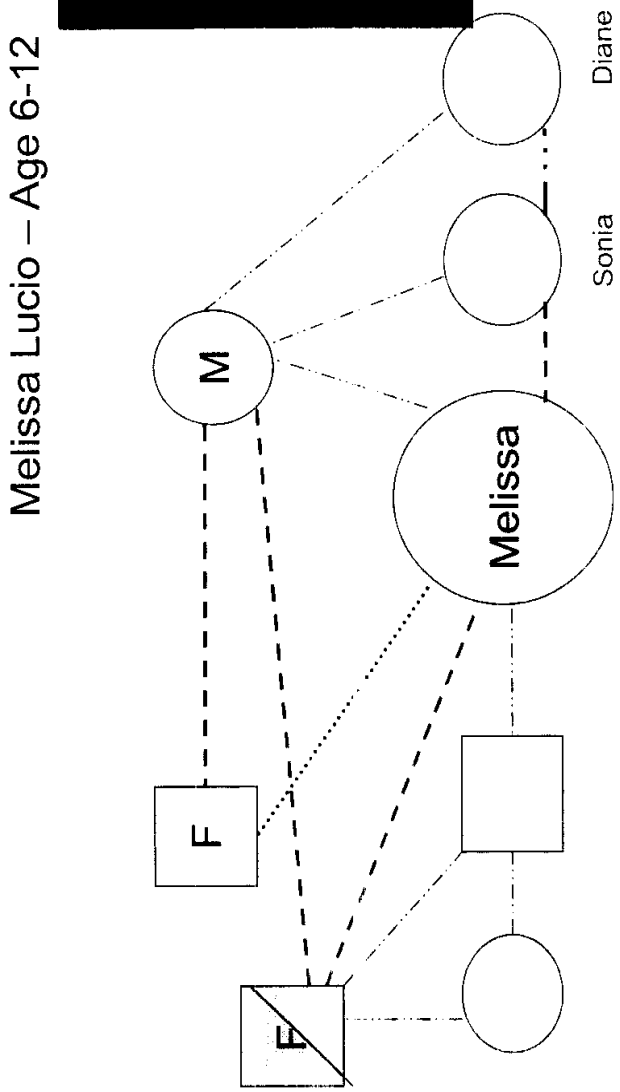
Violent/Abusive relationship

Strained relationship

PAGE 1

[redacted]

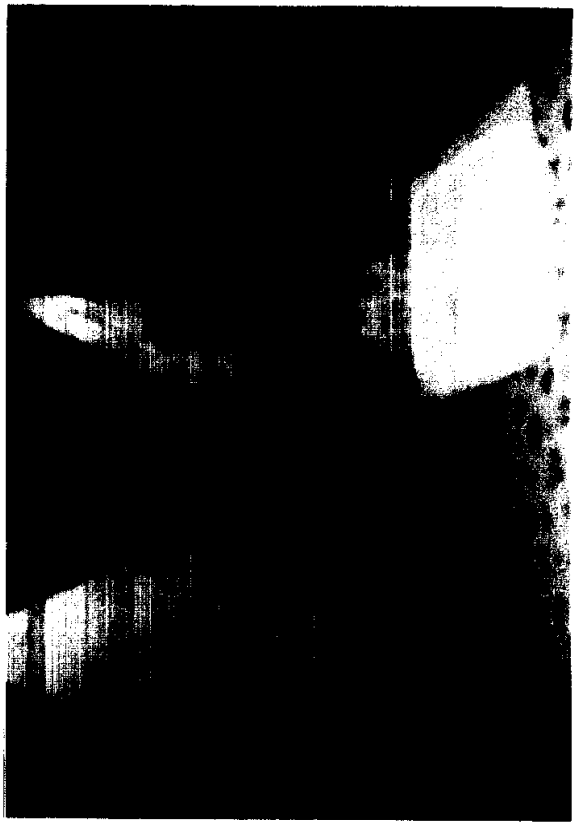
Melissa Lucio – Age 6-12



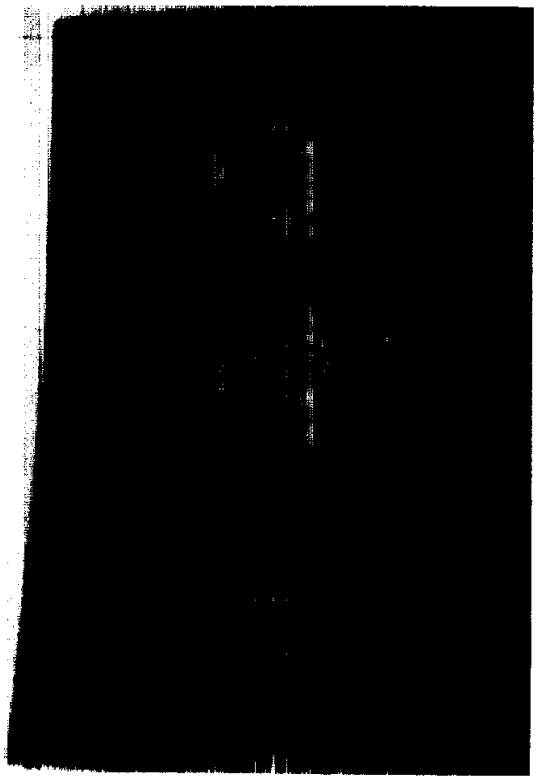
Melissa sexually abused. Step father with mother for two years. Mother did not believe/protect.
Melissa physically abused by Sonia.
All children witnessed the abuse suffered by mother.

PAGE 2

Functional relationship
No relationship
Violent/Abusive relationship
Strained relationship

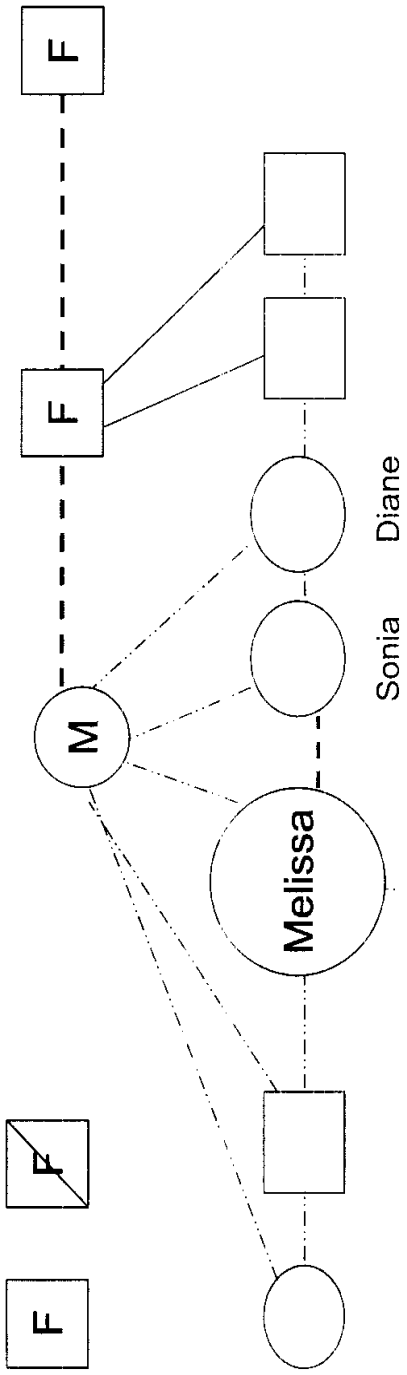


Melissa 8 (sisters birthday)



Melissa 6

Melissa Lucio – Age 12-16



Last 2 step fathers abusive to Mother not abusive to children
Melissa marries age 16.
Moves to Houston.

Functional relationship - distant

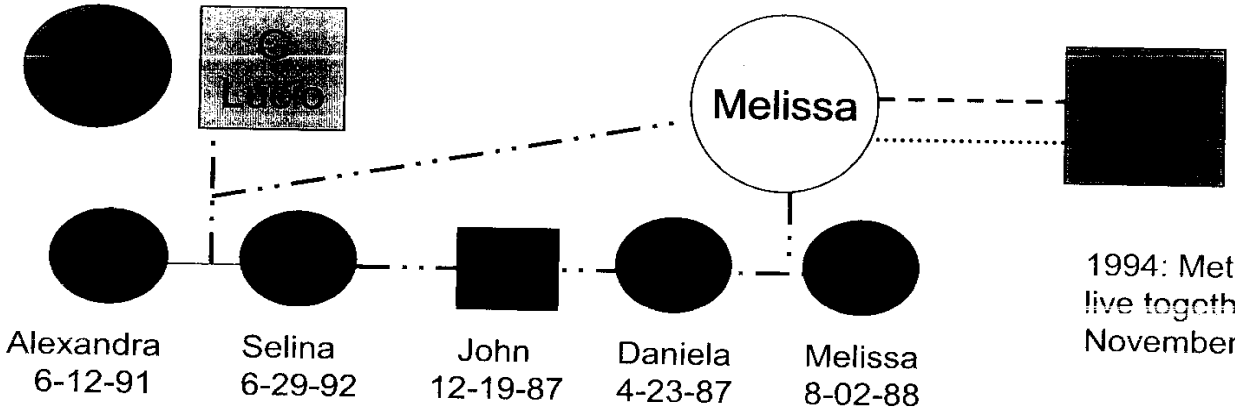
No relationship

Violent/Abusive relationship

Strained relationship

PAGE 4

Melissa Lucio – 1992-1996

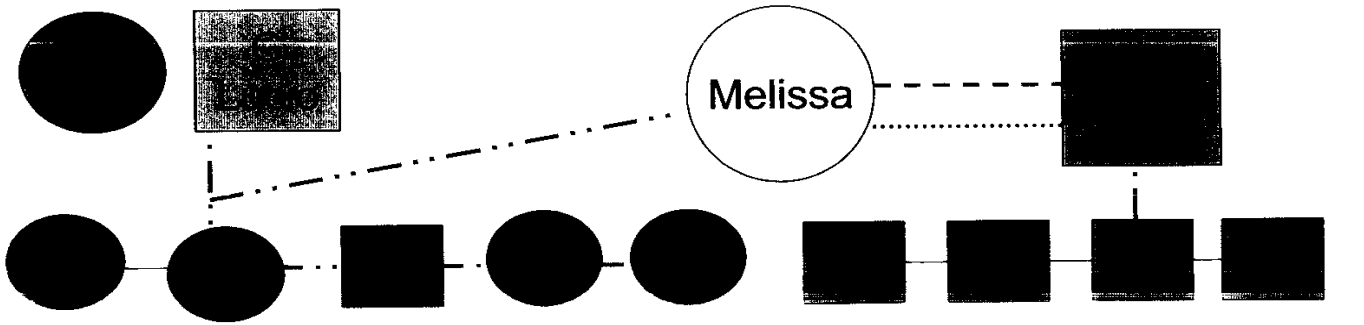


August 1994: Mr. Lucio abandons family.

———	Functional relationship
.....	Drug relationship
- - - - - -	Violent/Abusive relationship
—·—·—	Strained relationship

CPS: Neglectful Supervision 1995 (2), No action taken. No drug testing or notation of lack of food.
Cocaine use/abuse.
Insufficient food in home.
Children left unattended.
Melissa starts Norplant birth Control 1992. (side effects)

Melissa Lucio – 2001

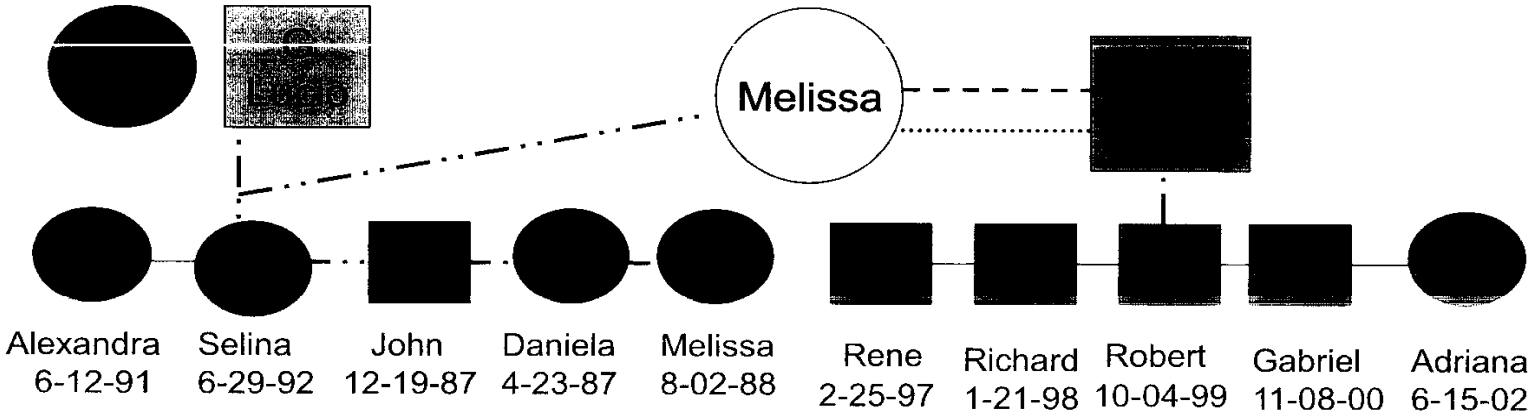


Children violent with each other, become angry and enraged. Fighting cussing, punching, biting

_____	Functional relationship
.....	Drug relationship
- - - - - -	Violent/Abusive relationship
—..—...	Strained relationship

11/2001 Physical neglect:"factors controlled"
12/2001 Physical neglect:"factors controlled"
Family homeless living in park, refused to go to shelter. School principal saw Mr. Alvarez hit Ms. Lucio. No action on first call. No children interviews, no drug testing, no contact with Mr. Lucio as biological father. Mr. Alvarez tells worker Ms. Lucio so depressed she is giving up.

Melissa Lucio – 2002



Children unsupervised, aggressive and violent with each other, dirty, and sexually acting out

_____	Functional relationship
.....	Drug relationship
_ _ _ _ _	Violent/Abusive relationship
_ . . _ . .	Strained relationship

5/2002 Neglectful supervision: Family Preservation (8/16/02-6/04/03)
Gabriel seen chasing cars and two minors girls sexually active. Daniela having sex with 20 and 30 yr olds, Melissa having sex with younger neighbors. Drug use by Ms. Lucio. Case closed: risk reduced.
No drug testing. Not clear if Daniela interviewed about sexual activity. Parenting contract made with no supervision or monitoring.

Melissa Lucio – 2003

Children unsupervised, using substances, violent. Bruising consistant.

5/2003: Physical abuse/sexual abuse Robert Alvarez: "factors closed/case closed" 6/2003 Neglectful supervision/ Physical Abuse baby born positive for cocaine. no action;
No collaterals contacted for investigation.
No contact with biological father.
No drug testing. No information on Lucio's substance abuse sessions. Lucio violated safety plan. No case consultation.

PAGE 11

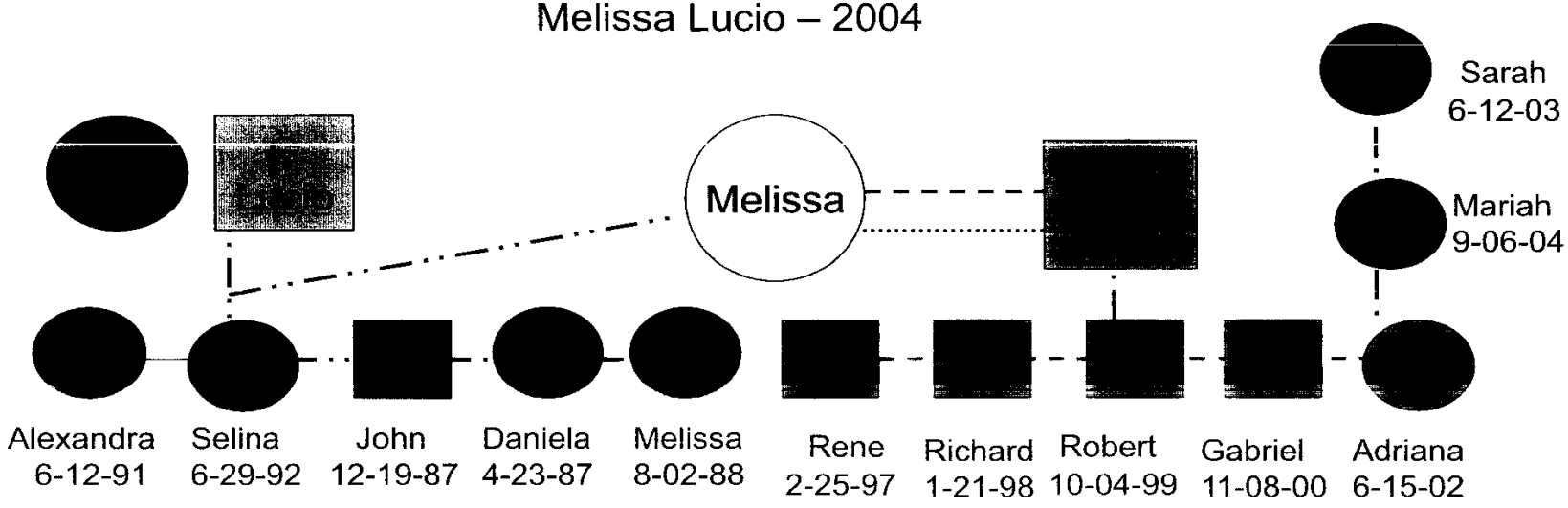
——— Functional relationship

..... Drug relationship

- - - - Violent/Abusive relationship

—·—·— Strained relationship

Melissa Lucio – 2004



Boys are all sexually acting out. This include between male and female Siblings and other children. All children in care bed wet. 3 Youngest urinate on selves during day, Children violent.

CPS case: 1/2004 neglectful supervision and physical neglect, "factors controlled" case closed. 8/2004 risk indicated. Mariah and Daniela.
No interviews with collaterals. Biological Father not contacted. No verification of 911 Call. Interview documentation incomplete. No courtesy check for children in Houston. Mr. Lucio admitted to alcohol use and not monitored.

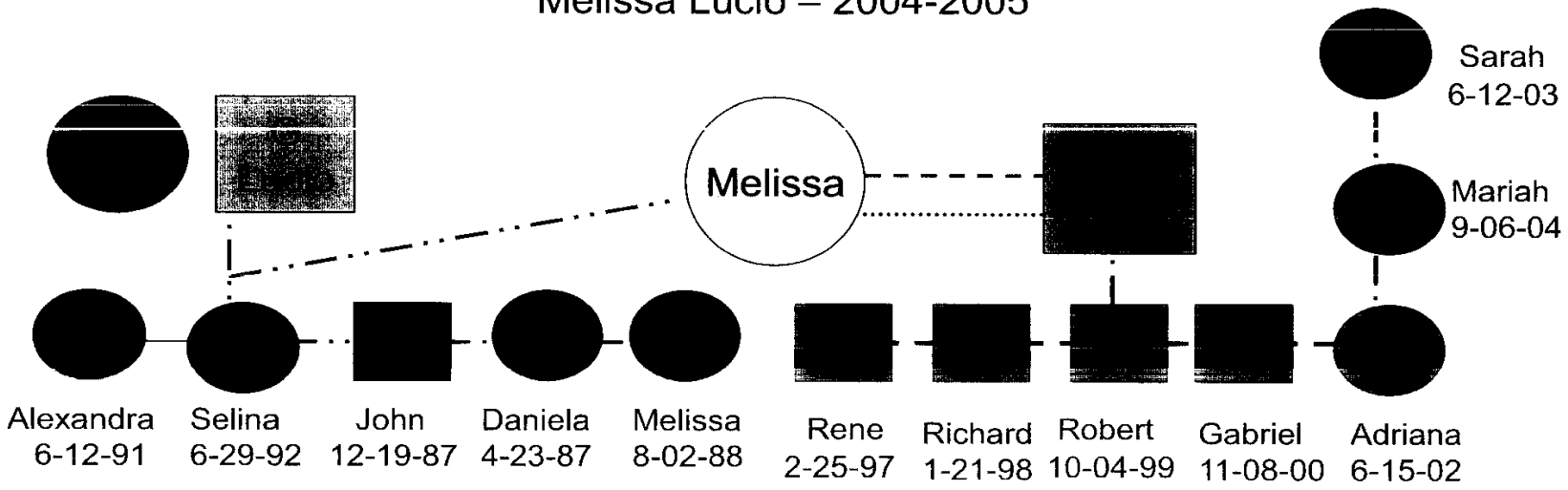
———— Functional relationship

..... Drug relationship

- - - - - - Violent/Abusive relationship

—··—·· Strained relationship

Melissa Lucio – 2004-2005



Foster parents note children violent with each other. Supervised visits indicate same. Parents cannot discipline or control children. The visits split children into 2 groups to help parents manage.

——— Functional relationship

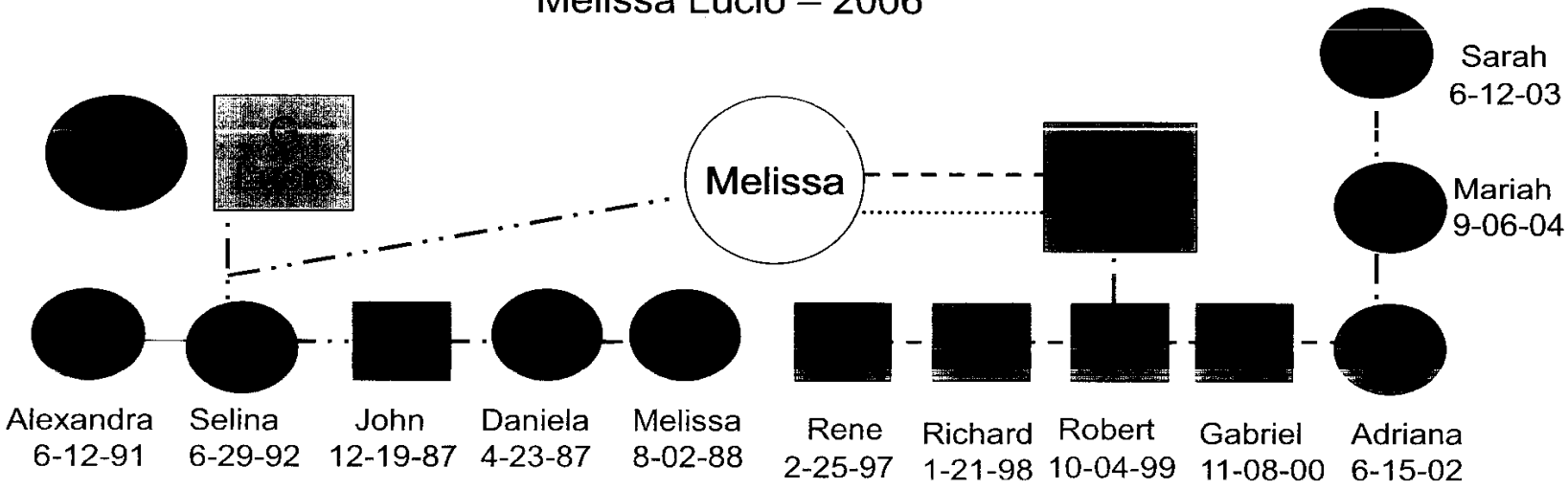
..... Drug relationship

- - - - - - Violent/Abusive relationship

—··—·· Strained relationship

CPS case: 1/2004 neglectful supervision and physical neglect, "factors controlled" case closed. 8/2004 risk indicated. Mariah and Daniela. Children removed.
No interviews with collaterals. Biological Father not contacted. No verification of 911 Call. Interview documentation incomplete. No courtesy check for children in Houston. Mr. Lucio admitted to alcohol use and not monitored.

Melissa Lucio – 2006



Children aggressive and
Violent with each other.
Behaviors consistent at
Home and in foster care

——— Functional relationship

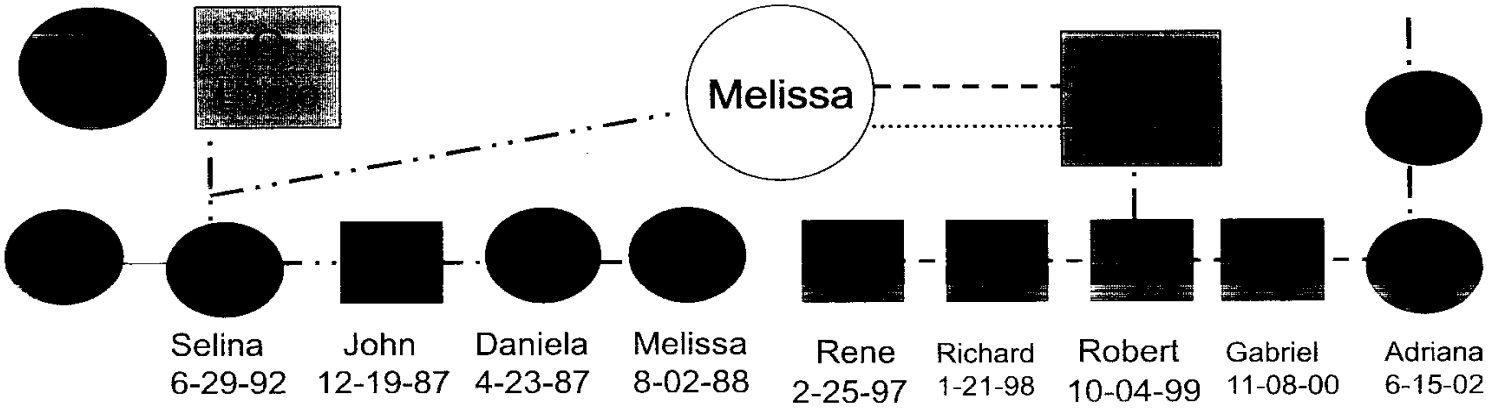
..... Drug relationship

- - - - - - Violent/Abusive relationship

—..—.. Strained relationship

CPS case: 12/2006 Unstable housing due To lack of paying rent, no electricity, Mr. Alvarez stealing furniture. 18 yr old (John) in home against safety plan. Supervised visits split because parents could not handle 8 children for 2 hours. Administrative closure of case. Family Assisted with rent. Both adults testing Negative on drug tests.

Melissa Lucio – 2007



All children returned at once. Within months family moving and had not furniture and utilities. Case not monitored.

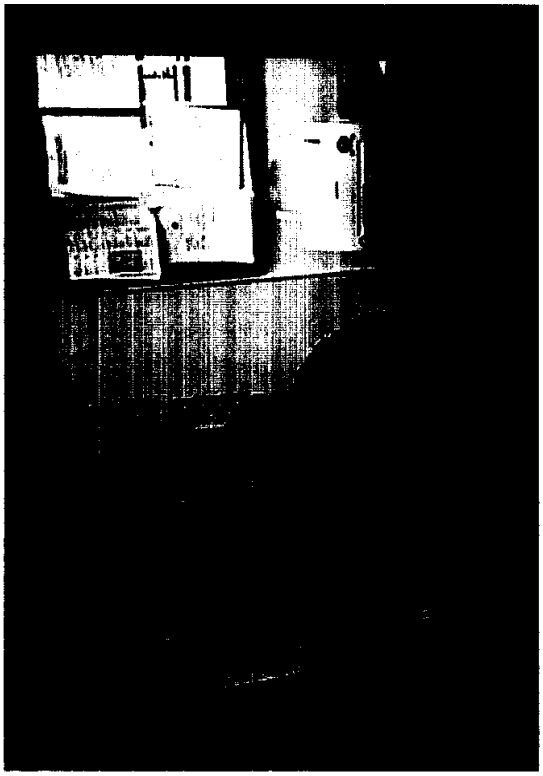
CPS maintains visits with mother and children in jail.

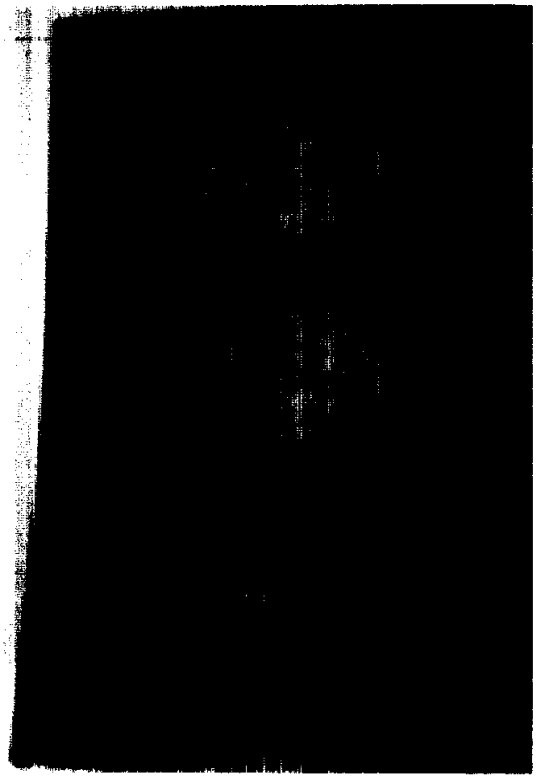
——— Functional relationship

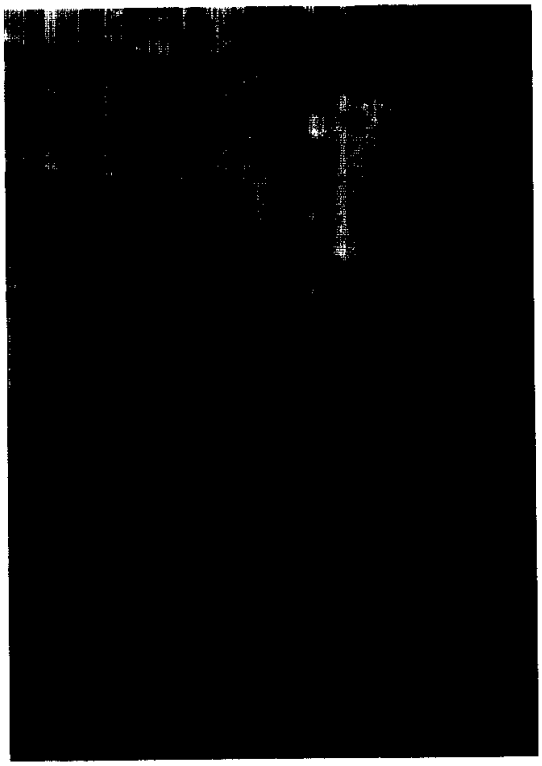
..... Drug relationship

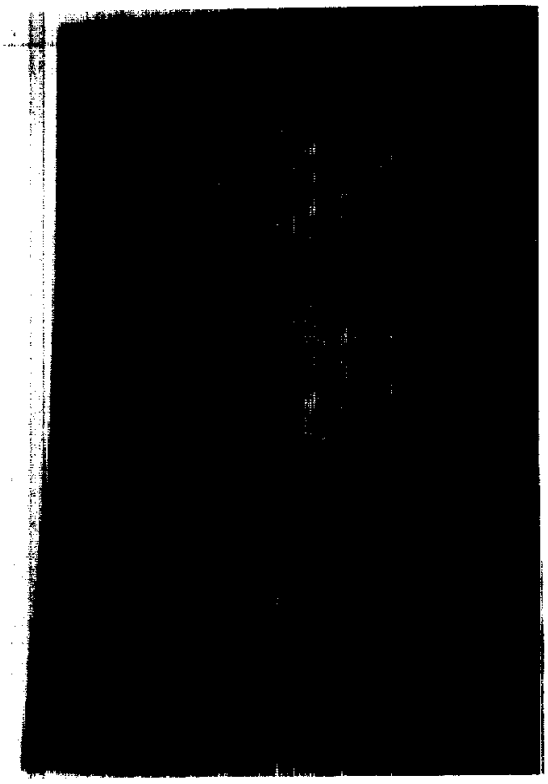
- - - - - Violent/Abusive relationship

—..—.. Strained relationship









Affidavit of Norma Villanueva

I, Norma Villanueva, state that the following is true and correct to the best of my knowledge:

1. My name is Norma Villanueva. I am over 21 years of age, can read and write the English language, and am competent to give this statement.

2. I am a Licensed Clinical Social Worker in the State of Texas. I obtained a bachelor degree in social work in 1980, and a master's degree in social work in 1985 from Our Lady of the Lake University in San Antonio, Texas. In addition to being licensed as a clinical social worker, I am also a Board Diplomate in Clinical Social Work.

3. I have worked as a mitigation specialist in capital cases since 1996. As such, I have consulted with capital defense counsel and conducted mitigation investigations. In cases involving Mexican nationals, I have traveled to Mexico to perform life-history investigations. I have worked on mitigation cases in Arizona, Texas, Oklahoma, and Missouri. I have also testified as an expert witness on mitigation issues in Texas. and Arizona state courts, and federal court in Texas and Missouri.

4. I was hired as the mitigation specialist on the Melissa Elizabeth Lucio case and visited with Melissa Lucio in jail with her attorney Peter Gilman on January 22, 2008. I was subsequently briefed on the case and the attorney's strategy. I was told to prioritize a review of the CPS case history to look for issues of errors and omissions in the management of the case.

5. On January 31, 2008, I had not yet received any CPS files and decided to speak with Mr. Gilman inquiring about the files, and also to speak about other mitigating topics to include family interviews. I was advised the CPS data was of top importance, and he was still waiting to have access to the files. I inquired about information to reach family members and was told by Mr. Gilman that family interviews were not a priority at this time. He committed to give me the family contact information he had in his file.

6. On February 14, 2008 I was furnished with the CPS documents. They were in several boxes. I picked up the boxes at Mr. Gilman's office and returned to my office. March and April were utilized to decipher the CPS data and compile sets of tables to organize information. The tables were furnished to Mr. Gilman, as the various sections were complete. I also interviewed Ms. Lucio in jail during this

time to obtain life history information and learned she was seeing a CPS hired therapist. I advised Mr. Gilman of this. It is noted here Ms. Lucio was unable to give contact information for family members. She stated she could not remember any telephone numbers. I asked Mr. Gilman during these two months for family contact information and was advised he (Mr. Gilman) did not want me to contact family yet. He wanted the CPS information to be the only task until completed. Mr. Gilman stated he did have family contact information and would be furnishing it at a later time.

7. On April 29, 2008, I informed Mr. Gilman of a comment in CPS notes stating that Mr. Alvarez was the last person to see Mariah alive. I also informed him of the well-documented physical abuse between the siblings by CPS. I asked for family contact information once again and expressed the crucial need to interview the family. I was advised to keep working on the CPS files as a priority. Mr. Gilman advised me he would let me know when I could proceed to contact the family and that he would furnish me their contact information at that time. I inquired about the possibility of interviewing Mr. Alvarez and was informed this would be difficult as he had another attorney and also pending trial.

8. May 15, 2008 we had a team meeting at Mr. Cordova's office. Mr. Cordova, Mr. Gilman, Dr. Pinkerman, and I were present. At that meeting, I reviewed information found in the CPS files to include the physical abuse between the siblings, the notation by a CPS caseworker citing Mr. Alvarez was the last one to see Mariah alive, and my concerns about a CPS hired therapist seeing Ms. Lucio in the jail. In addition, Dr. Pinkerman and I talked about battered women syndrome, and the effects on both adults and children who live in abusive homes and the role this played with Ms. Lucio and her children. We informed the team that family history and patterns would help to identify and utilize this. In addition, there was discussion regarding the need to find witnesses such as the school principal, Yvonne Montemayor, who witnessed Ms. Lucio being hit by Mr. Alvarez in the park near the school. Mr. Gilman discussed hiring a private detective to locate Ms. Montemayor and other witnesses mentioned in the CPS files. I was instructed to obtain information on the therapist, his ethical guidelines, and pathology ethical guidelines. I inquired once again about the family contact information and the need to interview family members. I was advised it would be forthcoming. I reminded the group that mitigation themes needed to be developed and investigated in order to fully present mitigation in the sentencing phase.

9. The beginning of June was spent obtaining requested information and state guidelines for Mr. Gilman as requested. I kept inquiring about the family contact information and the urgent need to interview the family members due to two factors. First, the CPS citation that Mr. Alvarez was the last person to see Mariah alive, and the issues of domestic violence. Mr. Gilman advised me the family

contact information would be forth coming. I reminded him of the urgency to clarify and investigate crucial mitigation themes and that time was running out.

10. On June 17, 2008, Mrs. Gilman requested I get information on the different CPS offices involved in the case so Mr. Gilman could send subpoenas to those offices to obtain updated information from CPS. This was during the jury selection process. I asked for family contact information once again and reminded the legal team of the importance of doing the family interviews.

11. On June 24, 2008 I interviewed family members of Ms. Lucio. The information from them corroborated the information in the CPS files to include the physical aggressiveness between the siblings. It was during this interview process that I met Alexandra, Mrs. Lucio's daughter. Alexandra stated she had been angry that day as Mariah was always crying and getting in between the other children while they were fighting and playing. Alexandra stated she "was the reason Mariah fell down the stairs". This interview ended in the evening. I attempted to call Mr. Gilman that night and left a message. The next morning, June 25th, I called Mr. Gilman and informed him of Alexandra's comment.

12. On June 26, 2008 there was a team meeting at Mr. Gilman's office to include Dr. Pinkerman. I informed Mr. Gilman again of my findings during the family interview that Alexandra stated she "was the reason Mariah fell down the stairs". I also had articles available regarding the effects of growing up in an abusive home. I was instructed by Mr. Gilman not to alert anyone or "make known" this information obtained from Alexandra to anyone, as it would result in criminal charges for this daughter. I questioned this as it served to change the entire trial for Ms. Lucio. I was instructed once again not to make this information know to anyone.

13. On June 29, 2008, Mr. Gilman requested articles on battered women's syndrome and the effects of domestic violence on the women and children. I emailed various articles that day to Mr. Gilman. I contacted the family once again to meet once again.

14. In July 2008, I continued to contact the family and conduct interviews. I was updating Mr. Gilman after each meeting. Issues regarding domestic violence, battered women's syndrome, and sibling-to-sibling violence was confirmed and given.

15. In preparing for my testimony, I developed a power point that included eco-maps, information form the CPS documents, and pictures of Ms. Lucio. I advised Mr. Gilman he could ask me questions about the family violence, sibling violence, and mental health issues as this information was all included in the power point slides. While on the witness stand, I was not questioned at all about the basic issues of domestic violence, sibling-to-sibling violence, or batter women's

syndrome so as to be able to give the jury details on these mitigating factors. In fact, I was not utilized during the sentencing phase at all. When I inquired about this with Mr. Gilman, I was advised my services were not needed.

16. During my testimony, questions were asked about incidents, which occurred while Ms. Lucio was incarcerated. The format of the questioning by the DA did not allow for full explanation giving the jury the impression Ms. Lucio's behavior was aggressive in the jail. Mr. Gilman did not address this during the cross-examination process at all.

17. I was not allowed access to the family by Mr. Gilman until the jury selection process had begun. This is not only too late into the case, it is not customary nor appropriate when a mitigation specialist is part of the team. In fact, I have never been denied access to the family by a defense attorney in any other case prior to this case.

18. The crucial information obtained on June 24 2008 during the family interview with Alexandra (Alexandra stated she "was the reason Mariah fell down the stairs") was not explored, examined, or given importance by Mr. Gilman. This information was simply disregarded and never utilized in any manner. I was advised I could not disclose this information to anyone. This was the first time I have been given crucial information during family interviews that was completely disregarded without any investigation at all by a defense attorney.

19. In order to conduct quality mitigation, a minimum of 9 to 12 months is needed. This is particularly true when individuals who need to be interviewed are in the care of the state and access has to be obtained. I was hired approximately 5 months prior to the jury being picked. Mr. Gilman made not effort to obtain extra time for the mitigation process. I was not given nor allowed to have contact with the family by Mr. Gilman, until the same month the jury was being picked for trial. More information could have been obtained to be utilized or mitigation purposes. Moreover, the CPS information could have been presented at trial by another individual allowing me the ability to present mitigation at the sentencing phase.

20. Mitigation that was not fully developed includes: interviews with Mariah's siblings, more in depth interviews with Ms. Lucio on the home life during her youth needed for the three generation biopsychosocial history, and interviews with Ms. Lucio's siblings.

21. Mitigation not utilized during testimony includes: the sibling-to-sibling physical abuse documented in CPS files, the history of domestic violence, Ms. Lucio's issues of battered women's syndrome, the effects of domestic violence on Mariah's siblings, the CPS documentation of Mr. Alvarez being the last one to

see Mariah alive, and finally the statement from the Alexandra that she was responsible for Mariah falling down the stairs.

I have read this affidavit. I affirm that it is true and correct to the best of my knowledge, and I so state under the pains and penalties of perjury.

Norma V. Villanueva, LCSW, DCSW

Norma Villanueva, LCSW, DCSW

Signed and subscribed before me
this 10 day January 2011

Queta D. Garcia

Notary Public

QUETA D. GARCIA
Notary Public, State of Texas
My Commission Expires 01-29-2012

My commission expires: 1-29-12

Respectfully Submitted,

Margaret Schmucker

Margaret Schmucker
Attorney for Defendant
Texas Bar No. 24030874

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Phone (512) 236-1590
Fax (512) 524-3479

CERTIFICATE OF SERVICE

I, Margaret Schmucker, certify that today, January 11, 2011, a copy of the brief for applicant and appendix were served by ~~certified mail~~ Federal Express, No. 8663 2191 0887 postage prepaid upon counsel for the State Rene Gonzalez and Lane Haygood at:

974 East Harrison St.
Brownsville, TX 78520
(956) 544-0849

Margaret Schmucker

Attorney for Applicant, Melissa Lucio

CERTIFIED BILL OF COSTS

THE STATE OF TEXAS §
§
COUNTY OF CAMERON §

I, Aurora De La Garza, Clerk of the District Courts of Cameron County, Texas, do hereby certify that the foregoing is a true and correct account of the costs accrued in the following entitled and numbered cause:

Cause Number: **07-CR-885-B-WR**

The State of Texas	§	IN THE **138th** JUDICIAL
VS.	§	DISTRICT COURT OF
MELISSA ELIZABETH LUCIO	§	CAMERON COUNTY, TEXAS

Given under my hand and seal of office in the **30TH** day of **MARCH**, 2011.

Aurora De La Garza
District Clerk

By: _____
CHRISTINA TUSA, Deputy

CLERK'S CERTIFICATE

THE STATE OF TEXAS §
§
COUNTY OF CAMERON §

I, Aurora De La Garza, Clerk of the District Courts of Cameron County, Texas, do hereby certify that the foregoing transcript contains true and correct original copies of all proceedings as to the Writ of Habeas Corpus in:

Cause Number: **07-CR-885-B-WR**

WRIT I

The State of Texas

VS

MELISSA ELIZABETH LUCIO

As they appear on file and of record in this office.

Given under my hand and seal of said Court of Cameron County, Texas, on **Wednesday, March 30, 2011**.

Aurora De La Garza
District Clerk
Cameron County

Christina Tusa
Deputy Clerk